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This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

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Industrial Solvents

—Methyl Alcohol; Benzine; Carbon Tetrachloride; Trichlorethylene; Benzol; Tetrachlorethane; Tetrachlorethylene; Carbon Disulphide; Tetraethyl-Ortho-Silicate; Diethylphthalate—

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OWING to the rapidity with which new solvents are placed upon the market, it is difficult for the industrial physician to keep pace with the literature dealing with the clinical phases of these substances after absorption through the skin or by inhalation.* As with refrigerants the fire hazard was given first consideration with little thought of the health hazard.

Having knowledge of the chemical formula and the boiling point, the toxicologist is able to state what may be expected of a new solvent. Solvents with a high boiling point such as acetonyl acetone, disobutyl ketone, and phorone, would be expected to be non-injurious to health, but could cause a dermatitis and ill health when absorbed through the skin

All volatile chlorinated hydrocarbons are poisonous. The great danger in the use of solvents lies in their ability to dissolve fats and lipoids alike in the body, in the skin, and in the nervous system. Some of these solvents gain access to the body by skin absorption, others by inhalation. Absorption by inhalation is the more dangerous as it comes in intimate contact with the blood and is carried to all parts of the body. The solvents may also have an irritating effect on the respiratory tract, laying one open to attacks of bronchitis and pneumonia. Some of the solvents may act very rapidly, while with others the effect is retarded. The symptoms of poisoning by vapor exposure may be acute or chronic, depending upon the nature of the solvent, the concentration in the air, and the length of time of exposure. The plant foreman is exposed to the solvent for short periods only, while his workman is continually exposed to it; the former, therefore, shows no effect and decries the thought of poisoning, while the latter, after some time, may show signs of chronic intoxication such as loss of weight, gastro-intestinal disturbances such as anorexia, constipation, occasional diarrhea, dizziness, irritability, asthenia, and loss of muscular coordination. The symptoms of chronic intoxication may be due to damage of the central nervous system, of the liver, and of the blood-forming organs.

Unnecessary exposure of the skin to any fat solvent, should be guarded against. Exposed arms and hands should never be immersed in a solvent. In the degreasing of small parts mechanical means should be employed. When the operation involves larger quantities of material to be degreased, exposure to the vapor may be lessened by closed systems. Consideration should be given to the selection of an appropriate solvent low in toxicity, and to the control of the concentration of the vapor in the air inhaled by workmen. The fire hazard of each solvent must be considered along with the health hazard. Many of the solvents have been thoroughly studied so that we now find proper safeguards employed.

Solvents may be divided into two general classes: volatile and non-volatile. Under the first are ethers, alcohols, benzol, toluol, xylol, carbon bisulphide, chlorinated hydrocarbons such as carbon tetrachloride, chloroform, trichlorethylene, trichlorethane, tetrachlorethylene, ethylene dichloride, naphtha, benzine and other petroleum distillates. Under the non-volatile are substances such as the vegetable and mineral oils, and the high-boiling ketones. The number of compounds used is legion. Doolittle[1] has classified over 100 solvents used in the lacquer industry.

Methyl Alcohol

THE toxicity of solvents was first brought to the attention of the medical profession in 1904 when Buller and Wood[2] collected 275 cases of serious poisoning by methyl alcohol, with 153 cases of

* Presented at the Nineteenth Annual Meeting of the Central States Society of Industrial Medicine and Surgery, at Springfield, Illinois, May 17, 1938.

blindness and 122 deaths. The toxic effect was reported by McFarlan in 1855. Baskerville[3] reported 64 cases of poisoning by inhalation of methyl alcohol. To eliminate poisoning by wood alcohol a denatured ethyl alcohol was introduced. This procedure, however, did not prevent many serious poisonings. This denatured alcohol was produced in enormous quantities—over 100 million gallons a year. This caused a marked decrease in the use of straight wood alcohol. When wood alcohol was synthetized from carbon monoxide, water, gas, and carbon dioxide, the sale of wood alcohol again came to the fore in 1926, and several hundred deaths were reported as due to its diversion from industrial purposes to beverage purposes.

Wood alcohol is a poison when inhaled, absorbed through the skin, or taken internally. Wood alcohol should be kept below one part per million in the air of work rooms. Blindness has occurred in a large percentage of the severe cases of poisoning. Nausea, dizziness, headaches, convulsions, and other symptoms may be seen similar to those following internal use. The air containing the solvent can be passed through a silica tube containing a heating element, the oxidation product tested for the presence of aldehydes by means of Schiff's reagent.

Benzine

ONE of the oldest solvents is benzine, known more familiarly as gasoline and naphtha. The latter are mixtures of paraffine hydrocarbons and comprise pentane (C_5H_{12}), hexane (C_6H_{14}), heptane (C_7H_{16}), and octane (C_8H_{18}). They are often confused with benzol (C_6H_6), which is not derived from petroleum. For a long time benzine was used in dry cleaning and was a prolific source of explosions and fires. It was sold under names such as petroleum naphtha, petrolic ether, gasoline, and Stoddard's solvents (a petroleum fraction, maximum boiling point 410°F., flash point 104°F.) and F-140. The first inhaling of benzine caused dizziness, the workmen complaining of "naphtha jags." Later it was discovered that this type of solvent caused blood dyscrasias. Hayhurst[4] was one of the first to acquaint us with the serious disturbances connected with severe exposures to the vapors from its use in industry. The petroleum distillates having a boiling point between 30°C. and 90°C. are used for motor fuels and are known as gasoline. Those with a boiling point below 150° C. are known as benzine, gasoline, petroleum, naphtha, etc. Substances with a boiling point between 70°C. and 150°C. are used chiefly as solvents. Petroleum naphtha is a generic term applied to refined, partly refined, and unrefined petroleum products of which not less than 10% distills below 347°F. (175°C.), and not less than 95% below 464°F. (240°C.). Petroleum spirits is a refined petroleum distillate with a minimum flash point of 70°F. (21°C.).

In the manufacture of varnish the petroleum benzine in some plants is added in open mixing tanks, the workmen inhaling enough of this solvent to become sleepy and dizzy. When they get out into the fresh air, they recover. People exposed like this are very apt to develop a leukopenia, a decrease in the red cell count and a lowered hemoglobin. As a degreasing agent it has been supplanted by solvents that do not have as great a fire hazard. Hayhurst reported a case of a man aged 48 who worked with this type of solvent as a degreasing agent. The solvent was composed of hydrocarbons similar to gasoline, 95% by volume distilling below 203°C., the residue being heavy mineral oil. Exposure to this solvent caused smarting and running of the eyes, burning sensation in nose and throat, headache, dizziness, anorexia, nausea, sometimes vomiting, "naphtha jags," nervousness, prostration, muscular twitchings, loss of strength, and gradual loss of weight from 138 to 121 pounds. The typical picture of chronic poisoning from petroleum distillate is loss of weight, worry, a feeling of "slipping" of physical and mental well-being. The blood picture in the Hayhurst case showed a severe secondary-anemia, 60-90% hemoglobin, red cells decreased 20-35%. There was a slight leukocytosis of 10,000 to 14,000; skin irritations, mouth infection, diarrhea, mental depression, and slowed mentality.

Frumina and Fainstein reported that rubber factory workers who were exposed to about 3 mg. of benzine per liter of air showed vertigo, excitability, and loss of sleep.[5] There is a higher incidence of functional neurosis (neurasthenia, hysteria, etc.); and bronchitis in female workers using benzine as a solvent.

Since some industrial installations of degreasing and dry cleaning processes employ only one or two men, these men are not given the attention that they should have. When an employee became ill it was thought that the work was too difficult for him and someone else replaced him. A hazard is not recognized, becoming a part of the expected labor turnover, until a claim is filed under the O. D. Act. The industrial physician should frequently consult with a chemist or an industrial hygienist concerning chemical hazards in the plant where he is employed. The chemist, however, is not always familiar with the toxic effects of a particular solvent; just recently my attention was called to a workman who nearly lost his sight while working with a new solvent

Wherever petroleum distillates are used, it behooves the industrial physician to investigate the working conditions more than he has in the past.

Carbon Tetrachloride

BECAUSE of the fire hazard with petroleum distillates carbon tetrachloride came into use. It was first used with mixtures of gasoline and naphtha to reduce the explosive properties of the latter. The naphtha mixtures required 30 to 70% of carbon tetrachloride to free them from fire and explosive hazards. Jones and Kennedy[6] show in their investigation that to eliminate the fire and explosion hazards to hydrocarbons ranging from hexane to octane, the final mixtures must contain at least 70% by volume of carbon tetrachloride. The "cracking process" above 1000°F. produces the

aromatic compounds: benzol, toluol, xylol, and other chemicals of this series; at temperatures below 1000°F. it produces olefines which are derived from unsaturated compounds such as ethylene, butylene, propylene, etc. It can be seen from this that petroleum naphthas may contain benzol, toluol, and xylol.

Carbon tetrachloride is a clear, colorless, mobile liquid. It has a characteristic ethereal odor, resembling that of chloroform. It is soluble in about 200 times its volume of water. It is miscible with alcohol, chloroform, ether, benzene, and with petroleum benzene, and dissolves most of the fixed and volatile oils. It has a specific gravity between 1.558 and 1.590 at 20°C. It distills between 76° and 78°C.

Carbon tetrachloride finds extensive use as a non-inflammable rubber solvent, as a fire extinguisher, as a delousing agent, and in medicine as an anthelmintic. It is found in some occupations such as those of airplane dope workers, rubber cementers, rubber cement mixers, degreasers in textiles, degreasing plants, dry cleaners, electroplaters, fire-extinguisher makers, lacquerers, lacquer makers, metal-polish makers, paraffine workers, perfume makers, refrigerator makers, mechanical parts degreasing, rubber workers, vulcanizers, and also in x-ray apparatus. Carbon tetrachloride has come into extensive use as a fire extinguisher, and also as a non-inflammable cleaning agent. The tetrachloride appearing on the market is often disguised by the addition of odorous substances or is mixed with small amounts of chloroform or petroleum oils.

Straight carbon tetrachloride has gradually replaced these mixtures in the cleaning and degreasing operations. You are undoubtedly aware of the dangers attendant on the use of this solvent in the home, as you may already have seen serious cases of illness and even death resulting from the inhalation of this solvent or its decomposition products.

Lampson and others subjected dogs to fumes of carbon tetrachloride at various concentrations and found that in 0.74% by volume, the animal became very restless and uneasy and there was an increased in salivation. As the concentration was increased, the coordination and balance were decreased and, when 1.94% by volume was reached, a quiet sleep was induced and a cessation of voluntary and involuntary movements.

All operations involving exposure to the vapor of this solvent should be done under a hood and away from free flames. The spilling of this solvent in workrooms has caused a number of cases of death and illness. Its use in fire extinguishers is a potential hazard, especially if used by the firemen to extinguish fires in limited spaces. I have seen several cases of serious illness and death from this source. Floor waxes containing this solvent should be applied when doors and windows can be opened so that an accumulation of the fumes in the room can be avoided. Concentrations of carbon tetrachloride should be kept below 100 parts per million. Fifteen milligrams per liter of air has been harmful to man. The gas is absorbed by all the organs. Robbins[7] found in the experimental work with dogs that the bone-marrow contained the most tetrachloride, with the liver containing the next highest. Small quantities were found in the blood, brain, kidney, lungs, etc. Practically all of the carbon tetrachloride was excreted in the lungs and hardly any in the urine.

Smyth and Carpenter[8] report that 1000 parts per million of carbon tetrachloride is safe for periods up to 30 minutes per day. The day's average, however, should not exceed 100 parts per million. This gives an odor which is barely perceptible. A very sensitive nose may detect 50 parts per million.

In reviewing the literature Smyth found 27 fatal cases of poisoning. I believe there are many more cases not reported which have occurred not only in industry but also in homes. Of 96 cases of acute and sub-acute poisoning Smyth found only 18 were non-industrial. This number, I again believe, is low, as in one degreasing department of a large firm in Chicago there were 18 casee in which a suit was instituted for damages. In one of these, which came to trial, the verdict was in favor of the defense. The other 17 never came to trial. In most cases an added resistance is established during a short rest from exposure. A few cases of idiosyncrasy are noted. This contrasts with benzene, where the injury produced is progressive even during periods of rest. Standard dry cleaning machinery keeps the maximal safe concentration more or less at a constant level.

In Davis'[9] experiment where he exposed subjects to 0.25 gm. per liter from three to seven minutes, there were nausea, vomiting, dizziness and sleepiness. Both the pulse and respiration were increased. An analysis of the urine after the experiment and two weeks later failed to show albumin, sugar or casts.

The plant physician should be very careful in the selection of persons who are to work with any chlorinated hydrocarbons. Those having nephritis, diabetes, myocardial degeneration, high blood pressure, and those using alcohol, should not be permitted to work with these solvents, for if any exposure occurs with a temporary layoff, the individual will certainly claim that he developed the nephritis or that his other conditions were markedly aggravated by the exposure. Every precaution should be used to prevent the inhalation of this gas, and the concentration should be kept below 0.01 to 1%.

The symptoms of carbon tetrachloride intoxication are: headache, hiccough, nausea, vomiting, diarrhea, pain and tenderness in the liver region, and jaundiced sclerae characterize the early stages; oliguria, urine containing albumin and casts follow next; then symptoms of uremia, increasing in severity as the suppression of urine becomes complete. Death is preceded by uremic convulsions. A toxic amblyopia and low blood sugar were reported by Wirtschafter.[10]

Intravenous injection of calcium gluconate and ingestion of glucose are recommended in the treatment of acute poisoning.

In 1876, Vitali devised a simple halide detector which involved the heating of a copper cone by an alcohol lamp. If any halogen is present, a blue-green copper flame is obtained. The intensity of the color obtained, is roughly proportional to the concentration of the halogen present. He was the first one to use a test such as this for the detection of mehyl chloride.

Trichlorethylene

TRICHLORETHYLENE is a chemical derived from ethylene by the replacement of three hydrogen atoms with chlorine. It is a heavy colorless liquid which volatilizes to a colorless, heavy, non-flammable, non-explosive gas with a characteristically pleasing ethereal odor similar to chloroform.

The liquid is approximately one and one-half times as heavy as water, having a density of 1.472 to 1.476 at 15°/4°C. Pure trichlorethylene boils at 86.7°C. (188.1°F.), but commercial grades have a boiling range of 86.0 to 87.5°C., indicating a relatively high purity. It freezes at —88°C. (—126.4°F.).

Its vapors are 4.53 times as heavy as air. The vapor pressure at 20°C. (68°F.) is 60 mm. of Hg. At normal room temperatures (20°C.–68°F.) air saturated with tri vapors carries approximately 0.027 lbs. of trichlorethylene per cubic foot of dry air. Concentrations as high as this are, however, rarely encountered under practical conditions of use.

Most commercial grades, however, show an alkaline reaction when shaken with distilled water to which an indicator such as phenolphthalein or methyl orange has been added. This is due to the presence in these grades of trichlorethylene of small quantities of organic materials known as stabilizers or inhibitors which are added to increase the stability of the material under average conditions of use.

The common industrial metals do not have any effect on the stability of trichlorethylene. However, prolonged contact with aluminum or magnesium, or their alloys, may result in the formation of aluminum or magnesium chlorides which act as catalysts accelerating the decomposition of trichlorethylene.

Although trichlorethylene is considered as non-flammable and non-explosive, at high temperatures it will decompose. The ignition temperature has been found to be 410°C. When sprayed into a gas flame or on to a red-hot metal surface, flashes of flame will be seen around the heated zone. If air containing trichlorethylene vapors is drawn into or comes in contact with open flames such as gas flames, arc or gas welding, the vapors will decompose with the formation of toxic and corrosive substances such as hydrochloric acid and phosgene. This type of decomposition will also occur if air containing trichlorethylene vapors is inhaled through burning tobacco such as lighted cigarettes, cigars or pipes. Exposed electric heaters or high pressure steam pipes may also cause this type of decomposition.

Obviously, therefore, open flames, welding and smoking in the vicinity of equipment using trichlorethylene should be prohibited. Gas-heated equipment using trichlorethylene should be equipped with suitable acid resisting flues or vent pipes so that the combustion products are safely disseminated outside of the building.

As a solvent for oils, greases and waxes, trichlorethylene is among the most efficient known, and has a very widespread use in a large variety of industries. Its chief uses are the removal of oil, grease and other lubricants from metal articles prior to finishing operations in the so-called solvent degreasing processes and the dry cleaning of garments and textiles.

Most of the reported cases of poisoning were due to inhalation of the vapors. Poisoning may occur through skin absorption followed by toxic symptoms. Furthermore, prolonged contact of the skin with the liquid solvent or its vapors may produce excessive dryness and subsequent cracking due to the extraction of the natural oils present in the skin.

Trichlorethylene is related chemically to chloroform and carbon tetrachloride, having a similar narcotic as well as an anesthetic action, but it differs from them in that it does not cause fatty degeneration of the liver. There is probably no cumulative action from small quantities or it would be evident in those who use it day after day for tri-facial neuralgia. However, there are serious chronic sequelae following acute attacks of poisoning. These may consist of anesthesia in the region supplied by the trigeminal nerve distributed over the greater part of the face and mouth and multiple neuritis.

A young man, 26 years of age, working for one year on a degreasing operation with an open tank of trichlorethylene became unconscious upon several occasions. Four weeks ago he was forced to stop working because of lack of feeling in his hands and feet. His history prior to working on the degreasing job was negative. He now has a well-developed case of muliple neuritis. A second young man, 27 years old, worked with trichlorethylene for two years cleaning furniture. He developed a gastric ulcer, and pains in his right lung which have persisted for five months. The x-ray of his chest was negative. A third young man had severe eye disturbances and was forced to stop work for several weeks. He returned to work but not on a degreasing job. The cases of polyneuritis, loss of reflexes, and lowered sensitivity of the skin are increasing in number. I believe this would be true of other chlorinated hydrocarbons.

Of more consequence is atrophy of the optic nerve leading to loss of vision with loss of corneal reflex of the eye. There are, however, some indiactions that this may be due not to the trichlorethylene itself but to certain impurities which until recently have normally been present in commercial grades of trichlorethylene.

Stuber[11] reported 284 cases of trichlorethylene poisoning, 202 of which were acute and 82 chronic. Of the total 25 were fatal, and in 17 of these there was loss of consciousness. In some there were symptoms of drunkenness or dullness and con-

fusion; in others there were general nervous symptoms and nausea and vomiting. One of the cases who died was held to have been a case of phosgene poisoning. Willcox[12] claims to have seen a case of jaundice from trichlorethylene.

Taylor[13] found that the concentration of pure ethylene in air of 0.5 and 0.4% trichlorethylene vapor produced deep anesthesia in rats, while 0.2% gave only slight effects.

The Underwriters' Laboratories report on trichlorethylene[14] gives the results of exposure of animals to vapor in concentrations of 0.6 to 1.0% by volume (2.0 to 3.4 pounds per 1000 cubic feet). Two minutes after the beginning of a five-minute exposure the guinea pigs breathed irregularly, and they coughed and rubbed their noses. Four minutes after the start of the test the animals showed considerable lack of coordination. The animals recovered within a day of the test and appeared to be normal during the remainder of the observation period. In the 30-minute exposure the animals appeared semi-conscious wtihin 20 minutes after the start of the test and were lying on the floor of the cage. Three guinea pigs removed from the room at the end of 30 minutes after the start of the test were semi-conscious and showed violent trembling, especially in the legs. All the animals appeared normal within 30 minutes after they were exposed to air but were very lethargic and weak. They recovered within a day after the test. Practically the same observations were noted with animals exposed for one hour.

In the two-hour test the animals were able to walk and eat within 30 minutes after they were exposed to air, but were very weak. They made a complete recovery within a day following the test. Where concentrations of 2.0 to 2.3% (6.7 to 7.7 pounds per 100 cubic feet) were used, they found that the guinea pigs were unconscious at the end of a two-hour period and showed occasional weak twitching movements. Two of the animals died within 20 minutes without regaining consciousness. The other animals appeared very lethargic and weak on the day following the test and showed some difficulty in breathing.

One of the animals which died following exposure to trichlorethylene vapor-air mixture for two hours was autopsied. The lungs were found to be hyperemic in general and contained innumerable petechial hemorrhages throughout. The upper left lobe was particularly filled with such hemorrhage. There was some edema. The cut sections did not produce any pussy exudate. The liver was hyperemic, but otherwise normal. The heart was engorged with dark clotted blood, but otherwise normal. The spleen was pale and yellowish in color and unusually soft. The stomach and intestines contained semi-fluid contents. The intestinal contents were slightly colored with blood. Upon opening the abdominal cavity, a definite hyperemia and moderate edema of all the viscera was noted.

Recovery from trichlorethylene anesthesia is generally complete but vertigo and dizziness sometimes occur. Striker, Goldblatt, Warm and Jackson[15] have investigated the use of this drug as an anesthetic and found no harmful consequences in any of the 304 cases examined. Kramer[16] reported no harmful after effects in any of the attempts to use it in cases of trifacial neuralgia. Stuber[17] included in her reports of chronic trichlorethylene poisoning damage to the liver, kidneys, optic and trigeminal nerve, ductless glands, sex organs, the skin and central nervous system. Plessner[18] reported injury to the trigeminus and subsequent apoplexy due to the specific action of trichlorethylene. Gerbis,[19] in 1928, and Kalinowski,[20] in 1927, have shown that the action was in all probability due to some impurity.

Because of the danger of formation of phosgene by the gas flame, gas-fired machines should be condemned unless equipped with suitable ventilation or flues so installed as to disseminate outside of the building all of the combustion products.

Stabilizers have been added to commercial grades of trichlorethylene to prevent their decomposition under normal conditions of use. However, some decomposition of the solvent may occur with the formation of hydrochloric acid. Industrial physicians have therefore found it necessary in some plants to frequently change men who work around degreasing units because of irritation of the throat and nose due to this decomposition product. In some plants attempts have been made to lower the amount of free acid liberated by the addition of a mild alkali, such as soda ash, to the degreasing tank. This, however, has no effect whatsoever on solvent vapors decomposed outside of the tank.

TABLE I.

COMPARATIVE TOXICITY OF CHLORINE DERIVATIVES OF THE ALIPHATIC HYDROCARBONS

(Relative lethal concentrations by volume in air if toxicity of CCl_4=1)

(Table arranged in ascending order of toxicity)

Methyl Chloride	CH_3Cl*	0.6
Tetrachlormethane (Carbon tetrachloride)	CCl_4	1.0
Trichlormethane (Chloroform)	$CHCl_3$	2.2
Perchlorethylene	C_2Cl_4	1.6
Trichlorethylene	C_2HCl_3**	1.7
Dichlorethylene	$C_2H_2Cl_2$	1.7
Pentachlorethane	C_2HCl_5**	6.2
Tetrachlorethane	$C_2H_2Cl_4$**	9.1

* WALLER, A. D.: *J.A.M.A.*, 53:9, 1919.

** HERRMANN, G., Inaugural Dissertation; Wurzburg, C. Fuchs, 1911.

See McNALLY's "TOXICOLOGY," p. 795.

The relative toxicity of trichlorethylene as compared to chloroform is 1.7. McCord[21] believes that 10,000 parts to 1,000,000 parts of air are definitely narcotic in man. He made a study with rabbits in which death occurred within two hours with concentrations of 5,000 parts per 1,000,000; in 28 to 41 hours with concentrations of 1,000 per 1,000,000, and in 19 days one death with 500 parts per 1,000,000.

"When trichlorethylene is discharged into the surrounding air in initial concentrations of the order of 1% by volume in the presence of flame

or hot objects, the resulting volatile products, if in unventilated or confined spaces, are lethal to guinea pigs for durations of exposure of the order of half an hour. When discharged into the surrounding air in initial concentrations of the order of 2% by volume under the conditions described above, the resulting volatile products are lethal to guinea pigs for durations of exposure of the order of five minutes. It is to be noted that the toxical products are exceedingly irritating and give definite warning of their presence."

Henderson and Haggard in their book, "Noxious Gases,"[22] give the relative toxicity of a number of chlorinated hydrocarbons in comparison with carbon tetrachoride, as shown in Table I (page 299).

Benzol

BENZOL is a colorless limpid liquid with a characteristic odor, boils at 80.2° C., having a specific gravity of 0.879. It is soluble to the extent of 0.07% in water.

The commercial varieties have been found to contain some toluene, xylene, olefins, paraffines, and carbon disulphide. It is somewhat soluble in alcohol, and is an excellent solvent for rubber, gums, resins, celluloid, and fats of all kinds. It is used extensively in the rubber industry, in varnish and paint removing, in gilding and bronzing, in spray gun paints, coal tar paints, and to a small extent as motor car fuel. It is found in a gasoline produced by the "cracking process' previously mentioned.

Although the dangers of benzol have been known for a long time, cases of poisoning still appear in Illinois and other states. Just recently I examined a young woman who had worked in a factory for eight years, taking bottle caps out of an oven. Her work also consisted of inspecting the caps taken from the drier; there was a rubber cement on these caps which contained benzol. The temperature of the drier was 130° F. The girls employed in this type of work did not wear masks. The patient stated that the caps would stick together and, in taking them apart, she could smell benzol. She was employed in this process all those years before becoming ill. After appropriate treatment however, she was able to return to work in another department.

The clinical picture is one of a progressive anemia, with a sense of malaise, fatigue and weakness, then bleeding from the gums and nose, followed by rapid decline, retinal hemorrhage, petechiae, hemorrhage from the mouth, stomach, intestines, and uterus, fever more or less intense, sometimes chills and delirium, and death from anoxemia and heart failure with pulmonary edema. Typically, the leucopenia should be a granulopenia, in some cases the polynuclears do fall as low as 10% of the leucocytes. If there is a simultaneous infection there may be enough active marrow tissue left to produce a response in the form of increased polynuclears[23] which cloud the diagnosis of benzol poisoning, as only a leucopenia was considered. The red cells usually do not present any abnormality, however, stippling and even nucleated red cells may be found. The ultimate result in clinical terms is an agranulocytic anemia, accompanied by an aplastic anemia. In one case in which the benzol was both inhaled and absorbed, the white blood cells were 950, red blood cells, 3,500,000, and hemoglobin, 60%. This is an example of a poison having an insidious and long-drawn-out onset with no pathognomonic signs whatever, until a sense of weakness compelled the patient to cease work.

Friemann,[24] in examining a large number of workmen handling benzol, found that there was a decreased vitamin C excretion. Normally there is 50 to 80 mg. up to 100 mg. excreted in an average 1000 cc. of urine, but in workmen handling benzol this amount is considerably decreased. The author concludes that benzol unquestionably has a marked influence upon the excretion of vitamin C, this sign being pathognomonic in early cases of benzol intoxication. It should be borne in mind that benzol, like other solvents, can be absorbed through the unbroken skin. Experiments have demonstrated that benzol penetrates the skin faster and in larger quantities than does benzine.

Smyth states: "Toluene being less volatile than benzene is also less toxic. Xylene is quite toxic, but less volatile than toluene, therefore less hazardous. Neither seems to be readily absorbed through the skin in toxic amounts."[25]

Sartorius and Sudhues[26] report that it is possible to expose rabbits several hours daily over a period of one and a half years to benzol-air mixtures of 13.6 to 27.2 mg. per liter of air without noteworthy changes. This is probably true of many other poisons, our best data being derived from the examination of human beings exposed to known amounts of poisons as seen in industry.

All people working with benzol should have a periodic blood count and a periodic examination of the urine by the sulphate test. This latter test shows the absorption of benzene before the usual blood changes occur, and should always be made while the subject is at work, never after a rest period such as over Sunday, because the sulphate content changes during that period. Low values in a group of workmen exposed to benzol are an indication that additional ventilation is necessary.

The urine sulphate ratio is the amount of the inorganic sulphates in the urine divided by the total sulphates. This ratio is a measure of the amount of benzol vapors inhaled by the individual tested. When it is below 50% it is a warning that additional ventilation is necessary; below 25%—definitely dangerous. Where the blood examination shows a leukopenia, the workman should be removed from the source of benzol. Patients having symptoms such as bleeding from the nose or mouth, should be immediately hospitalized and then treated by fractional transfusions of 50 to 100 cc. of blood daily. Injections of pentnucleotide, liver extract, and large doses of iron and ammonium citrate by mouth should be given. X-ray of the long bones should be used for its stimulating effect upon the bone-marrow.

Tetrachlorethane ($CHCl_2$-$CHCl_2$)

TETRACHLORETHANE, sometimes called acetylene tetrachloride, is used in airplane "dopes" or varnishes, with which the wings and other parts of airplanes are painted. It is also used in the manufacture of non-inflammable films, lacquered goods, and artificial silk. Tetrachlorethane, prepared industrially by the action of chlorine upon acetylene, is a heavy, colorless, oily liquid with a boiling point of 147°C. (296.6°F.) and a specific gravity of 1.614. The vapor is about six times heavier than air, a very important factor in connection with the dangers from its industrial use. It is non-inflammable, and has a sickening sweet odor suggestive of chloroform. It is an excellent solvent for certain types of cellulose esters, resins, etc.

Zangger saw seven cases of poisoning among workers in a chemical industry. Jungfer and Wilcox[27] reported cases of poisoning from the use of "dope" in airplane factories. The outstanding symptom was jaundice. Grimm[28] reported 18 cases of poisoning in Germany, and in England at least 70 cases of jaundice, with 12 deaths. Schibler[29] reported three fatal cases of acute yellow atrophy of the liver due to this solvent. They were all employed in a small shoe factory. The solvent was used with a glue employed in the factory.

The early symptoms begin with abnormal fatigue, discontent general nervousness, and loss of appetite, which are followed by nausea, vomiting and dizziness. The blood at this stage of poisoning is striking, and is of aid in diagnosis. There is a progressive increase of large mononuclear cells, often reaching 40%; there are many immature large mononuclears, a slight elevation in the white cells and a progressive but slight anemia. If the patient was removed from the exposure when the jaundice was slight, complete recovery occurred, but slowly. If the jaundice is severe, the patient will not recover, as reported by Schibler in his cases.

Tetrachlorethylene

CARPENTER[30] exposed 136 rats for various intervals to 70, 230, 470, and 7000 parts per million of tetrachlorethylene. These were continued over a seven-months period, eight hours daily, five days weekly, the maximum exposure being 150 days or 1200 hours; no deaths were recorded. Growth was not markedly impaired; fertility was stimulated rather than inhibited. Periodic blood and urine chemistry revealed no indications of damaged organs or altered function; no effect upon blood picture. At autopsy, however, slight changes were found in the liver, kidneys, and spleen; other tissues were normal.

It was noted that 50 parts per million produced only a definite odor; 500 parts per million produced only slight discomfort; and 1000 parts per million caused light inebriation in 45 minutes. Hence a concentration of 100 to 500 parts per million of tetrachlorethylene vapors is thought safe for daily exposures not in excess of 40 hours weekly.

Carbon Disulphide

EXPOSURE to this chemical is found among ammonium salt makers, artificial silk makers, asphalt testers, carbon disulphide makers, celluloid makers, cementers, (rubber shoes), cement mixers (rubber), dry cleaners, dryers (rubber), enamelers, glue workers, insecticide makers, match factory workers, oil extracters, paint makers, paraffine workers, putty makers, reclaimers (rubber), smokeless powder makers, sulphur extracters, tallow refiners, and vulcanizers.[31] Pazen[32] was the first to call attention to it as an occupational poison, although the poison was known in 1796. It boils at 46.5°C. It has a specific gravity of 1.262. It burns with a bluish flame, yielding carbon dioxide and sulphur dioxide. When passed through a red-hot tube it yields carbon tetrachloride and sulphur monochloride. It dissolves in its own weight of sulphur at 38°C.

Two thousand cc. of the gas per cubic meter of air are fatal within five to ten minutes exposure. Five hundred cc. per cubic meter of air are endurable from one-half to one hour exposure. Concentrations over 300 parts per million in the air for several hours cause symptoms of acute poisoning. Workmen should not be exposed to concentrations over 0.1 mg. per liter.[33] The symptoms of acute poisoning are characterized by circulatory disturbances, pallor headache, throbbing of temples, palpitation, fainting, and drowsiness. Chronic symptoms may develop in a few weeks or months or after several years of work where the concentration is low. Disturbances of smell, taste, and sight are common. Organic nervous symptoms are common and consist of partial and complete paralysis, especially of the extensor group of muscles leading to wrist drop, steppage gait, and later, contractures. The patient cannot read because of disturbances of vision, disturbances of color fields, and retrobulbar neuritis. When removed from exposure, recovery may be very slow. Treat symptomatically. The characteristic odor of this gas is a sufficient indicator of its presence.

Quarelli[34] describes a clinical syndrome produced by carbon disulphide, where the corpus striatum, which is especially rich in lipoids, is the seat of the selective action. The syndrome is characterized by a Parkinsonian-like manifestation. It began with a slight muscular hypertonia followed by tremors and a polyneuritis, involving especially the sciatic nerve. There is sexual frigidity, loss of libido, and impotence. The tremors are also present at rest, similar to those occurring in paralysis agitans.

All these symptoms are suggestive of an extrapyramidol involvement of the central nervous system as a result of chronic disulphide poisoning.

Gordy and Trumper[35] reported six cases of carbon disulphide poisoning. Two of the patients showed corneal anesthesia, two complained of wavy vision, two had retrobulbar neuritis and, in one acute case, illusions were present. Cohn[36] found 30 cases of psychosis which developed in one viscose plant. All these cases occurred in the

rayon industry in which a Federal survey is now being made.

Tetraethyl-Ortho-Silicate

KASPER, McCORD, and FREDERICK[37] have called our attention to the toxicity of tetraethyl-ortho-silicate.

Tetraethyl-ortho-silicate, $Si(OC_2H_5)_4$, is a limpid water white liquid, boiling at 165.5°C. Its density is 0.933 20/20 and its vapor pressure relatively low. Air saturated with ethyl silicate vapor at room temperatures (25°C) will contain approximately 17 milligrams per liter of the vapor. The liquid has a characteristic odor, unobjectionable to most persons. In common with most esters, ethyl silicate is only sparingly soluble in water or aqueous solutions, but itself is an excellent solvent for fats, oils and similar high-molecular-weight organic compounds. It is miscible with alcohols, esters, and ethers.

Ethyl silicate is used as a vehicle and solvent in paints, and lacquers, as a stone preservative, in heat resistant coatings, as a cement or bonding agent for refractories and other materials, and as a strengthening or hardening agent in sand molds, brick, textile products and the like. Small quantities of ethyl silicate are used in dentistry for the sealing of dental surfaces.

From other experiments they found that ethyl silicate was highly toxic. When introduced intravenously into rabbits, 0.2 ml. per kilo of body weight caused death within one hour. The minimal lethal dose for rats, when administered intraperitoneally, is approximately 0.06 ml. per 100 grams of body weight; with rare exception, death takes place within four days. An interesting

TABLE II.

Name of Solvent	Use in Industry (Solvent for)	Toxic Effects	Autopsy Findings
Ethyleneglycol	Dyes — Supplementary agent for lacquers	Slight irritation of mucous membranes	
Carbon disulphide	Rayon industry—solvent for rubber, sulphur, resins, and wax	Polyneuritis, ataxia, tremors, mental confusion, visual disturbance, anemia	Hemorrhages and congestion, fatty liver. Focal necroses in brain
Carbon tetrachloride	Chemical industry—Rubber—Fumigation—Cleaning and dyeing	Chronic abdominal pain, vomiting, headache, vertigo, coma, edema, urinary suppression, jaundice, hepatitis, secondary anemia. Polyneuritis, nervousness	Congestion of viscera. Parenchymatous degeneration of kidneys. Hepatitis. Fat necrosis. Jaundice. Several cases mentioned in literature
Ethylene chlorbromide dibromide and dichloride	Chemical industry—water-proofing preparations — "Ethyl" gasoline — Dye intermed. Fumigation. Oils, waxes, cleaning	Irritation mucous membranes. Headache, vertigo, nausea, vomiting, coma, anemia	Congestion and edema of lungs. Degenerative changes in kidneys. Conjunctivitis. Pharyngitis. Bronchitis.
Trichlorethane	Dry cleaning	Acute narcotic poison. Skin lesions like burns. Secondary anemia	Congestion of lungs and other viscera. Diminution of hemoglobin. Fatty changes in liver
Trichlorethylene	Dry cleaning—Solvent in rubber	Multiple neuritis. Action on C.N.S. Paralysis of sensory portion of N.V. Secondary anemia	Capillary injuries of cerebral vessels
Tetrachlorethylene	Dry cleaning—Waxes, resins, oils—Anthelmintic	Nausea, giddiness. Vomiting, slight conjunctivitis, headaches	Not stated
Tetrachlorethane (acetylene tetrachloride)	Production of cellulose acetate	Acute yellow atrophy of lover. Chronic emaciation, mono-nuclearosis, hemolysis, secondary anemia, basophilic stippling, jaundice	Acute yellow atrophy of the liver and fatty changes in other viscera
Acetylene tetrabromide	Liquid gauges—Separation of ores		
Monochlorbenzene, orthodichlorbenzine, trichlorbenzene	Chemical industry—Sulphur dyestuff, metal polish, varnishes, dry cleaning, fumigation	Usually by absorption through skin. Irritation respiratory tract, anemia, hemoglobinuria, leucopenia with relative lymphocytosis	Not stated
Propylene dichloride	Dry cleaning—greases. Fumigation	Headache, vertigo, lacrimation, anemia	Marked anemia
Methyl acetate	Solvent for cellulose, resins, oils and fats	Irritant. Narcotic action. Headaches. Changes in blood. No human deaths reported	Generalized congestion. Diminished blood coagulation. Sub-mucosa hemorrhages
Ethyl acetate	Cellulose, nitro-cellulose, oils, fats	Anorexia, loss of weight, blood changes. Increased r.b.c. and hemoglobin. One death reported	Congestion of viscera and eyes. Liquid blood in vessels. Petechiae. Pungent odor in opening body
Amyl acetate	Celluloid, resins, oils and fats	Drowsiness, irritation of throat. Tachycardia, tinnitus, nausea, gas-irritation, coughing	Parenchymatous degeneration of kidneys. Secondary anemia and leukocytosis. Fatty changes in liver
Propyl acetate	Same—	Irritant mucous membranes. Narcotic action	Hyperemia and congestion especially respiratory tract and viscera—Fatty changes in liver. No blood changes
Butyl acetate	Same—	Irritating. Conjunctivitis, narcotic action. Anorexia, loss of weight, headache, abdominal pain, tendency to anemia	Congestion of viscera, hemorrhages of mucous membranes and skin. Absent postmortem clotting of blood. No death recorded in man
Benzyl acetate	Nitro-cellulose	Tremors, headache, severe anemia, also ataxia	None specific except blood changes.
Diethylphthalate	Nitro-cellulose. Used also as softener	Irritation mucous membranes. Paralysis C.N.S.	Produces hemorrhagic nephritis fatty changes in liver. Decrease in Hb. Increase r.b.c. Relative polynucleosis
Methylglycol acetate	Same—	Slight irritative and narcotic action	Destroys hemoglobin and r.b.c. and is also leukotoxic
Acetine	Same—	Less irritating than acetic and esters. Narcotic	Destruction of hemoglobin and r.b.c. No change in leucocytes

feature of every research was that the ethyl silicate acted chiefly upon the pulmonary tract. Pulmonary hemorrhage associated with ruptured capillaries, may be demonstrated within 10 minutes after appropriate intraperitoneal injection. In the animals which survived the effects of the primary action of the ethyl silicate, a definite acute nephritis was found.

Many solvents may cause contact dermatitis. Poisonings have occurred from the chlorinated naphthalenes, as perchlornaphthalene. It is produced as a substitute for resins, and rubber, and for the impregnation of textiles and insulating material. It is poisonous when inhaled, causing headache and a feeling of weakness, with uncertain gait. On the skin it causes folliculitis, similar to that seen in chlorine acne.

Of the hydrogenated naphthalenes, tetra- and deca- hydronaphthalene (tetraline, $C_{10}H_{12}$, and decaline, $C_{10}H_{18}$) are useful solvents for resins and fats. When used industrially, or in newly painted and polished rooms, they may produce headache, nausea, vomiting, and irritation of the conjunctiva and of the mucous membranes of the nose and throat. Inhalation of tetraline leads to the excretion of an extraordinary grass-green urine owing to the oxidation of the tetraline to tetralol.[38]

One solvent seldom mentioned is diethylphthalate. It is used in the manufacture of nitrocellulose, as a solvent for oils in the perfume industry, and, during the Prohibition period, it was used as a denaturant for alcohol. In contact with the skin it causes a severe dermatitis. It has been demonstrated that the glycols, especially diethylene glycol, when taken internally are very toxic. The glycols are oxidized partially to oxalic acid and I believe, due to that, oxidation would be a source of intoxication through the skin.

Since time will not permit even to mention the 400 different solvents now in use I have included a table of some common solvents described in this paper with their uses, toxic effects, and autopsy findings. (See Table II, page 302.)

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Recognition of Early Pulmonary Tuberculosis in Industry

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THE important community health problems are, incidentally, the important health problems of industry. This applies particularly to pulmonary tuberculosis. While there has been a marked reduction in the importance of the tuberculosis problem, both for industry and the community, in the last 30 years, it still retains its importance from the standpoint of disabilities which are long and serious. Thirty years ago there was little or no provision for discovering or caring for tuberculous individuals. There were no clinics, there were no sanatoria, nor was there routine physical examination for any purpose and particularly that for employment. The practice of making routine physical examinations only applied to a few such selected occupations as train service on railroads and for service in the army or navy.

Without attempting to evaluate the various factors which have entered into the reduction of the death rate from tuberculosis in Illinois in the last half century from 250 or more per hundred thous-

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and to less than 60 per hundred thousand, we do know that our knowledge of the situation and our management of cases is greatly improved. At the beginning of the Twentieth century records available seemed to indicate that the incidence of tuberculosis among those employed in industry was approximately the same as that of the community. While the community death rate in Illinois from pulmonary tuberculosis is something less than 60 per hundred thousand at the present time, the tuberculosis death rate in many of our large industrial groups is less than one-fourth that of the community rate. The case rate of tuberculosis in industry is only slightly in excess of the death rate in the community. In recent studies made of large industrial groups, the death rate from pulmonary tuberculosis during the year 1937 was 14 per hundred thousand. The incidence during this same period, of known cases of pulmonary tuberculosis, was 80 per hundred thousand.

These figures correspond very closely to the general conception of those who have studied the tuberculosis problem, that for every known death from pulmonary tuberculosis there are from five to seven active cases in the community. It seems, then, that while we still have the problem of pulmonary tuberculosis among those employed in industrial establishments, the problem is not nearly so great as it was 30 years ago and that there has been a very definite change for the better by reducing the industrial rate from approximately equal to that of the community rate to less than one-fourth of the community rate.

Because of the fact that a lot of loose statements have been made regarding the unfavorable relation between employment in industry as such and the unfavorable effect upon the tuberculosis rate, it might not be out of place again to repeat statistics regarding the change in the tuberculosis death rate in large industrial centers. It might be well to note especially in this connection that the reduction in these large industrial centers in the tuberculosis rate has been more rapid than that in the states in which the industrial centers are located.

TUBERCULOSIS MORTALITY PER HUNDRED THOUSAND

	1900	1930	
City		City	State
Chicago	157.8	56.9	74
St. Louis	196.3	59.1	83
Detroit	113.0	68.5	68
Louisville	211.5	60.2	108
Pittsburgh	133.7	57.7	69

Five years ago there was great excitement in this country about silicosis and silico-tuberculosis. At a public meeting in Washington two years ago no less a person than the Honorable Secretary of Labor stated in public that an investigation made for the Department of Labor had shown that at least 500,000 people in the United States were about to become disabled or were going to die of silicosis or silico-tuberculosis. The civil courts were being flooded with damage claims numbering thousands of cases and aggregating claims of millions of dollars for compensation. Since this hysterical period many health surveys have been made by competent authority. Recent statistics of thorough and careful surveys, at which time adequate x-ray chest plates have been made, in places like Chicago, Milwaukee, and more recently in New York State, show conclusively that while there are certain occupations in which silicosis may be produced and silicosis or silico-tuberculosis result in disability or death, the number of workmen involved and the number of jobs with serious potential hazards are very much smaller than was thought even two years ago. A survey of a large group of factory employees in Chicago was recently made. A large percentage of all those examined had been, or were, occupied in the so-called dusty trades. Counting everything that could be possibly interpreted as some evidence of lung change due to dust exposure, either clinical or x-ray, or both, counting every stage from earliest to the most advanced, the total number found of any degree, irrespective of whether there was disability or not, was less than 2%.

Beginning about 25 years ago, some of the larger industrial establishments started to examine applicants for employment before they were assigned to jobs. The fundamental purpose of this pre-employment examination was to assist in the choice of a man physically qualified for the particular job which he wished to do. Shortly after this pre-employment examination was established the practice of making periodic examinations of those already employed was also established. Following the lead of those who started this work 25 years ago it has become almost a universal practice in all except the smaller industrial establishments to more or less regularly carry out these two procedures. Within the last year or two these examinations have been made much more carefully than was previously done. The usual type of examination at the present time includes not only urinalysis and blood pressure, but also an x-ray chest plate. Needless to say, with the improvement in technique, the work now is much more accurate and satisfactory.

There has been considerable discussion about these physical examinations, both on the part of management and labor, and in some places objections have been raised to making any physical examinations at all. The objectors very frankly stated that they thought the theory of these examinations was excellent, but in some places these examinations were used for some other purpose than determining physical and mental fitness and because of this applicants for employment and those already employed were unfairly treated. The subject of mental "fitness" was broadened into mental "attitude"—attitude as exemplified by religion, politics and trade unionism. The obvious comment is that any physical examination that is made must, *first*, be well done and, *second*, the doctor making this examination must not abuse his privilege nor allow the knowledge which he has gained to react to the disadvantage of the man who is seeking employment or who is already em-

ployed. Honesty and fairness must characterize this work or it cannot continue.

THERE are two or three interesting points in the plan of procedure in making physical examinations. It is a well known fact that wherever there is a rapid increase in the number of employees of an industrial establishment, due to the pressure of increasing business, it regularly happens that a considerable percentage of those put to work, subsequently, within the next few days drop out because they do not like the work or because they or the employer find they are not qualified for the job which the employment department thought they were. It has been argued that there is a large amount of wasted effort in making pre-employment examinations of everyone before they actually go to work. In fact it has been the practice in some industries to wait a few days or a month or so before making this examination, the argument being that if the plant physician waits this short interval he only has to examine those where it is reasonably certain that they will remain on the job.

It is my personal opinion that such a procedure is not fair either to the employee or to the management. It is very embarrassing and frequently the subject of considerable argument, if a man has been allowed to work a few days or a month, finds that he likes the job and can perform it to the satisfaction of his foreman, and then is physically examined and it is found that he has some physical defect or disease which disqualifies him for employment. It is believed that reasons for rejection are hard enough "to take" under any circumstances, and that in fairness to the man he should be told before he actually goes to work, whther or not he is physically qualified.

The plan of periodic physical examination of those already employed has been the subject of a great many comments. Without going into an elaborate argument about the reasons for the plan, my personal opinion is that all employees, whether they are executives or common laborers, should have a complete examination no less often than once in three years. Supplementing this, those who are in responsible positions, those who manage or direct others, should be examined at least once a year. Then in addition to this, all employees who are subjected to a known occupational hazard of any degree should be examined more often than once a year. If employees are subjected to a known poison, then according to some of the state laws, it is necessary to check the physical condition of this group once a month. In a word then, the schedule of examination should be, *first,* everybody at least once in three years; *second,* those in responsible positions once a year; *third,* those who are subjected to occupational hazards of any degree, frequently enough to keep a constant check on the effect of the exposure.

As previously stated, records of disability and death, because of tuberculosis, in large groups of industrial employees, have shown a disability rate of about 80 per hundred thousand and a death rate of 14 per hundred thousand employees. This does not include those who have evidence of tuberculosis, either old or recent, but who have not as yet developed disability. On careful examination, including chest x-ray, of large numbers of those applying for work, there were some clinical or x-ray signs of pulmonary tuberculosis in 2.4%; of those already employed there were clinical or x-ray signs of tuberculosis, but no symptoms, in 1.9%.

If we compare the cases where there is a clinically active pulmonary tuberculosis with the number which are found to be working who have x-ray or physical signs of some inactive tuberculosis, we find that for every clinically active case there are 25 who are regularly working and in spite of having x-ray or physical evidence of some old pulmonary tuberculosis they do not have any evidence of active disease. Experience has shown that only a very few of those of this group who are classified as having x-ray signs of some pulmonary tuberculosis, as discovered in this type of routine periodic examination, ever developed an active pulmonary tuberculosis. This fact is very important because there has been an outspoken opinion on the part of many managers, on the part of some industrial physicians and even fellow employees, that if tuberculosis is discovered in such an x-ray examination sooner or later active tuberculosis will develop and such an individual is always a potential hazard. Such an attitude has resulted disastrously for a good many men who are well placed and working well within their physical limits. This phthisiophobia should have prayerful consideration wherever a group of industrial physicians are gathered together. We must learn to distinguish between the man who has active pulmonary tuberculosis with positive sputum, an active pulmonary tuberculosis but without positive sputum, and "x-ray" pulmonary tuberculosis where the individual is in good physical condition and working at a job where means the difference between economic independence and insecurity. As physicians, irrespective of whether we have any interest in industrial work, we should ever keep in mind that the best preventive for tuberculosis is a regular job in a fairly decent working place with a regular pay check and the possibility of a decent place to live and regular meals.

THE disposition of the cases of tuberculosis—speaking of them in the all-inclusive term—whether active, quiescent, or entirely asymptomatic, should be given careful thought. I think we all can agree that where a man has an active tuberculosis with a positive sputum, he is a sick individual and should not be passed as an applicant for any job, and if he is employed he should stop work both for his own sake and for the safety of fellow workmen. An applicant, who from physical examination and x-ray is thought to have a tuberculosis which is not completely arrested, even if there is a negative sputum, should usually be placed in the same classification as the proved active case. A man who is employed, however, may frequently be allowed to work under supervision at his usual occupation, or some modifica-

tion of his usual occupation, without endangering his own safety or that of his fellows. An applicant who has an old inactive tuberculosis should not always be rejected for employment. Many such individuals can work at some self-supporting occupation with considerable benefit to themselves, their families and the community. Such an individual, however, should not be given employment which calls for excessive muscular effort nor should he be employed where he is exposed to a known occupational hazard. If such an individual is already employed and he has demonstrated that he is able to remain without symptoms at his usual occupation, it is seldom if ever necessary to disturb him unless he is in a known hazardous occution in which it might be possible for his old, latent disease to become reactivated by continued occupational exposure.

We now come to the problem of the employee who was compelled to quit work because of active tuberculosis and after a period of effective treatment at home or in a sanatorium is now ready to return to some regular work. It used to be taught that such an individual should only work out of doors—should seek an occupation in which he was more or less constantly in the open air. This idea is usually absurd, and most often it results in subsequent breakdown of the individual. If one stops to think, the exposure to the weather with the frequent and sudden changes in temperature and humidity and the laborious character of the work usually available in an outside job—the combination is anything but a satisfactory one for the tuberculous individual. The old cry used to be for a light outside job. Observation has shown that about the only light thing about an outside job is the pay. The experience of all of us who have dealt with cases of tuberculosis for years has been that the returned tuberculosis case does better at the job which he knows; the job for which he has been trained, possibly by long years of experience, the one that he does with the least effort and, incidentally, pays him the best wages. It is possible that his old job must be modified somewhat, but in view of modern standards of hours per day and days per week we must not fail to remember that the time off duty each day is usually 16 hours, and in addition to this there are usually two full days each week in which there is no work.

This should convince us that unless there is something inherently wrong with the old job the ex-sanatorium patient, if he is able to work at all, can get by very well if he limits his activities to the working hours alone. Various attempts all over the country have been made to return the individual with an arrested tuberculosis tc an especially selected job. All sorts of attempts have been made in various parts of the country and most of them have not proved very satisfactory.

On the other hand, it is true that any doctor who is familiar with the problem finds that he has quite a large percentage of the returned cases going along very well, year after year, on their old jobs.

IF WE wish to understand all the factors incident to the tuberculosis problem in industry we must not forget the determining factors in the development of any case of tuberculosis, under any circumstances. In spite of the fact that there is some evidence that adults occasionally acquire a primary infection after reaching adult life, it is still true that the great majority of all positive cases of pulmonary tuberculosis can be traced back to exposure in childhood. It is still true that in 100 consecutive cases of active pulmonary tuberculosis there is a positive history, in at least 50% of the cases of a direct exposure to a tuberculous mother, father, brother, sister, or some adult in the immediate family. We must not forget that living and personal habits have a great influence in determining whether or not a comparatively slight, old, latent infection becomes reactivated. We must also remember that working habits, not the working place or the occupation, have an important bearing. For example, in our medical schools the tuberculosis rate among students who have been carefully selected, is much higher than that in the community. The explanation for this high rate among medical students is not that they have any unusual exposure in their clossrooms or in their clinics, but modern educational practices in this field are so intense that medical students habitually, and over a long period of months and years, fail to get the adequate rest that young individuals require. There are very few jobs in which there is an exposure that is directly responsible for the activation of a tuberculosis, but most any job might be the determining factor. It is not the job itself, but the way that it is done that is important.

We have had a great deal of publicity on the question of industrial exposure as the determining factor in the development of tuberculosis. There has been a lot of "scare-head" publicity as to the enormous numbers of men who are subjected to such an exposure. Sober thought and careful investigation have shown that the importance of this type of industrial disease exposure has been greatly over-estimated.

Perhaps the outstanding factor in the development of tuberculosis is the economic status of the individual or his family. If a group of individuals have fairly decent jobs and work under fairly sanitary conditions and get a fair regular wage, it means that the majority of the individuals in this group will live regular lives in decent surroundings and their food, clothing and shelter are adequate. A group of individuals such as this usually has a very low tuberculosis rate, irrespective of the type of industry at which they are employed. If, on the other hand, a job is irregular, the working place is not very sanitary and the pay is the minimum, any group, depending on such jobs for a livelihood is bound to live poorly in unsatisfactory surroundings. We use the words susceptible and immune and yet no one knows exactly what susceptibility or immunity is. We ordinarily think of the colored race as being especially susceptible to tuberculosis. This is pos-

sibly true, but only in a measure. The most important factor in this race susceptibility is that such individuals are at the bottom of the economic scale and jobs are irregular and the pay poor. They live miserably, and all of the problems incident to health, because of such living, are emphasized. Even the so-called more immune will have a high tuberculosis rate under like circumstances.

I suppose all of you have been tremendously interested in Drolet's paper in the *American Review of Tuberculosis* for February, 1938, on the trends in tuberculosis. He shows by very comprehensive statistics for this country and England that the trend in the incidence of tuberculosis has been steadily downward. This means that the problem of tuberculosis in industry and in the country as a whole is gradually becoming less of a problem, and that in the future it will not be as difficult as it was in the past. Incidentally, however, in his paper he shows conclusively that where there are bona fide cases of active tuberculosis the results of treatment, irrespective of where or how, have not been very satisfactory. In other words, the hopeful sign is steady reduction in the number of cases and not what happens to those who actually develop the disease. Progress has come by prevention and not through treatment. The problem today is still important, but no longer of major importance. Any industry that has a trained medical staff and a fairly comprehensive plan for medical supervision can expect a steadily decreasing tuberculosis rate. The enormous number of routine chest plates that are being taken at the present time allow us to judge the relative importance of individual cases and the value of the contributory factors in the development of tuberculosis and how best to modify or control these factors. Knowledge of how to prevent tuberculosis in industry has come through persistent study of the employees and working places and their occupational hazards. The job is not yet finished, our methods of elimination and control have proved satisfactory; they should be continued.

Section on Railway Surgery

THE SECTION ON RAILWAY SURGERY, of the Southern Medical Association will hold its Annual Meeting in Oklahoma City, Oklahoma, November 15-18, 1938. DR. GEORGE A. TRAYLOR, of Augusta, Georgia, is Chairman of the Section; DR. OLIVER B. ZEINERT, of St. Louis, Missouri, is Vice-Chairman; and DR. JAMES W. DAVIS, of Statesville, North Carolina, is Secretary. The program of this meeting, subject to certain additions, is as follows:

1. "Chairman's Address," by DR. GEORGE A. TRAYLOR, Augusta, Georgia.

2. "The Treatment of Fractures of the Hip," by DRS. LAWSON THORNTON and CALVIN SANDISON, Atlanta, Georgia.

3. "Abdominal Distention and Intestinal Obstruction Following Skeletal Injuries," by DR. THOMAS G. ORR, University of Kansas, Kansas City, Kansas.

4. "Railroad Surgeons, as I Know Them in the Communities They Serve," by MR. JAMES J. DONOHUE, General Claims Attorney, Louisville & Nashville Railroad, Louisville, Kentucky.

5. "Eye Examinations in Railroad Service," by DR. H. vH. THATCHER, Stevens Building, Portland, Oregon.

6. "The Diagnosis and Treatment of Mastoid," by DR. B. C. DYER, Atchison, Topeka & Santa Fe Railway, Topeka, Kansas.

7. "Head Injuries," by DR. A. W. ADSON, Mayo Clinic, Rochester, Minnesota.

8. "Improved X-Ray Technique in Studying Knee Joints," by DR. E. C. HOLMBLAD, 28 East Jackson Boulevard, Chicago.

The profession is invited; the meeting, as the program indicates, will be of profound interest to *all* physicians and surgeons, as well as those engaged in railroad work.

Industrial Exposure and the Liver

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THE modern industrial worker finds himself called upon to make a great variety of complex bio-chemical and physiological adjustments in order to cope successfully with the environmental conditions under which he must work.* These abnormal conditions are, many of them, quantitatively as well as qualitatively, very different from those which characterized man's natural environment, and played a determining role in conditioning his biological evolution through the ages. Exposure to the elements, malnutrition, inadequate housing and lack of proper sanitation were perhaps among the principal factors of stress in the primitive environment. While many of these still exist to a greater or less degree, there has been added to them exposure to innumerable toxic substances in the form of poisonous and irritating dusts; irritating and asphyxiating gases and fumes; exposure to extremes of temperature and humidity not ordinarily met with in nature, etc. When one considers that the present industrial environment dates back to the industrial revolution—less than 100 years ago—and has developed in a more or less hit-and-miss fashion as a mere incident to subsequent technicological advances, the effective physiological responses of the body to the many new demands made upon it are truly remarkable.

From our knowledge of the subtle effects upon the metabolic processes of such elements in our

* Presented at the Industrial Hygiene Session of the Ninth Annual Convention of the Greater New York Safety Council, April 21, 1938.

daily routine as diet, fresh air, physical activity and the like, one can readily appreciate how many alterations in bio-chemical activity may be initiated by the absorption of poisonous substances or substances which seriously compromise the oxygen supply to tissue cells.

In all of these metabolic activities the liver holds a key position and is thus a vital factor in the worker's adaptation to his working environment.

The liver is concerned primarily with the metabolism of carbohydrates, proteins and fats. It stores reserves of glycogen which can be called upon by tissues as they require it. It appears to be responsible for maintaining the normal blood sugar level. Diaminization of amino-acids resulting from protein metabolism with the production of urea is another of its important functions. The bile which it excretes into the gastro-intestinal tract is essential for the digestion of fats as well as for the utilization of vitamin D. The liver converts carotin—the precursor of vitamin A—into vitamin A. It detoxifies many potentially poisonous substances. These are only a few of its typical activities.

Since the detoxifying functions of the liver are the ones which appeal particularly to the imagination of the industrial hygienist a word may be said with reference to them. Whether the substances are endogenous and come to the liver as the toxic by-products (or intermediary products) of normal metabolism, or are exogenous and come to it from without the body, the liver appears to have the power to oxidize or conjugate these substances in a great variety of ways and so provide for their subsequent excretion by the body in non-toxic or relatively less toxic form. Thus, benzol is conjugated by the liver with the formation of organic sulphates; cinchophen is oxidized and excreted as oxycinchophen. Alcohol, camphor, avertin, aromatic acids and many other substances are converted by the liver into glycuronates and excreted as such in the urine. The liver is thus capable of initiating a great variety of detoxifying chemical reactions, many of which are not fully understood as yet. Its importance as a chemical laboratory for the body can be best appreciated perhaps when one considers how large an organ it is compared with other organs in the body.

Up to a point the liver apparently succeeds fairly well in protecting the body against injury by toxic substances. When such injury, however, begins to interfere with normal function, one may get a secondary accumulation of the toxic by-products of normal metabolism—which the healthy liver ordinarily disposes of—in addition to the toxemia produced by the substance responsible for the initial damage. Because of the liver's enormous reserve powers and its great capacity for healing and tissue repair, serious interference with function does not usually occur until the pathological changes in the liver have become very extensive. For this reason it is not uncommon at autopsy to find that preceding complete histological disorganization of the liver—such as one gets in acute yellow atrophy, for example—resulting in the death of the patient after a very brief and stormy illness, the liver had apparently suffered a succession of gross injuries over a considerable period of time; each of which in turn had been repaired with a fair degree of success and without having given any very definite clinical evidences of the pathological processes which were in progress during this time.

THERE are a number of substances, industrial and otherwise, which appear to be especially toxic to the liver.

Among the anesthetics: chloroform and the rectal anesthetic, avertin, are the ones most commonly thought of;

Among the drugs: arsphenamine and cinchophen, or atrophan;

In industry: phosphorus, cadmium, possibly arsenic, manganese and copper, and various of the halogenated hydrocarbons—the better known among these being carbon tetrachloride, some of the chlorinated naphthalenes, dichlorethylene, trinitrotoluol, tetrachlorethane, etc.

Arsine, hemolytic serum, and other substances responsible for severe blood destruction may also cause liver damage indirectly through the strain put upon the liver to rid the body of excess cellular debris and waste products. Substances like carbon monoxide which cause an anoxemia are not generally thought of as liver poisons, and yet it is well known that liver cells are very sensitive to oxygen deficiency and are readily injured by oxygen lack. The same is true of many other toxic substances which are not ordinarily classified as liver poisons. Thus, for example, the question as to whether or not such a well known substance as lead causes injury to the liver is still being debated back and forth—chiefly because its toxic effects manifest themselves principally in other parts of the body, and injury to liver cells, unless very extensive, is extremely difficult to evaluate.

Because an understanding of the precise way in which specific substances disturb liver function would be expected to provide information as to general toxicity, many attempts have been made to appraise and measure these activities by means of selective liver function tests. Unfortunately, however, the great diversity of liver functions; the fact that one may be disturbed without necessarily involving the others; the enormous reserve powers of the liver, and its success in the regeneration of cells even after gross injury has been done, have all tended to minimize the value of such tests. That liver damage may exist in the absence of positive liver function is well known. On the other hand, excessive blood destruction, the presence of mechanical obstruction to bile channels, or other abnormal conditions entirely unrelated to impairment of liver cells may give results in liver function tests similar to those which are found in diseases of this organ. Interpretation of such tests, therefore whether positive or negative, may be very unsatisfactory. For this reason no at-

tempt will be made at this time to list them, or discuss the many functions which they are designed to measure excepting to say that the various tests are selective in character — each having as its purpose the appraisal of a single relatively well-defined liver function.

A word should be said, however, with reference to a most imaginative and practical contribution along these lines recently made by Yant and his co-workers—a method for estimating the extent of benzol absorption of workers in terms of changes in the relative concentrations of organic and inorganic sulphates in the urine. This test provides a simple chemical measure of exposure based on an understanding of the normal response of the liver to the absorption of this substance.

Normally, the inorganic sulphates constitute 85 to 95% of the total sulphates—the remaininig 5 to 15% being organic or so-called "ethereal sulphates." Absorption of benzol by the body is believed to result in its oxidation in the liver to phenol and phenol derivatives, which, in turn, are conjugated with sulphate-ions to form organic sulphates at the expense of the inorganic. Yant found that this decrease in inorganic sulphates is qualitatively related to the severity of the benzol exposure, and that normal relations are soon re-established when exposure ceases. The urine sulphate test thus provides an interesting index of absorption rather than toxicity; but in so doing it also provides practical information as to excessive absorption well in advance of toxicity, and so constitutes an invaluable aid to the prevention of benzol poisoning.

The urine sulphate test is not, strictly speaking, a liver function test, since it is essentially a measure of normal chemical activity. Nevertheless any departure from the normal response might give a clue to liver damage. Thus Yant suggests that with a liver poisoned by carbon tetrachloride one might not get the expected increase in organic sulphates associated with benzol absorption. This in turn suggests the further question as to whether a liver so damaged might not perhaps fail to make even the ordinary conjugations in a normal manner with the result that the concentration of organic sulphates in the urine might drop below the normal 5 to 15% level—where benzol plays no part in the picture.

I BELIEVE that the future of intelligent and effective medical prevention and control of industrial diseases depends, more than anything else, upon the building up of just such information on pre-clinical aspects of disease as Yant has provided for benzol. We must develop in so far as possible ways and means of measuring and appraising bio-chemical and physiological responses of the body to absorption of toxic substances well in advance of toxicity before recognizable clinical disease entities make their appearance. Since the liver plays such a fundamental role in these chemical adaptations of the body, a continued study of liver response to toxic exposure would seem to be one of the most fruitful fields for research directed toward the prevention of industrial diseases.

Within the time at my disposal it is impossible for me to even touch upon the many clinical and pathological aspects of liver diseases due to intoxications. A few practical points, however, in the early detections of these conditions, and the intelligent handling of persons who are exposed to known liver poisons might profitably be pointed out.

It is important to recognize, at the outset, that there appears to be an enormous difference in individual susceptibility to these so-called liver poisons. Large numbers of persons in the South have been treated with carbon tetrachloride for hookworm, and only relatively few have suffered untoward results. Certain individuals have died of acute liver atrophy after chloroform or avertin anesthesia and yet, relatively speaking, they have been very few indeed. The same holds true in industry. Workers exposed to carbon tetrachloride or other chlorinated hydrocarbons are very numerous, but the mortality or even morbidity due to liver injury has always been very low, so far as one can determine.

If one were able to ascertain susceptibility of individuals in advance of exposure the whole problem of industrial disease prevention would be greatly simplified. Incidentally, it has been possible by means of the urine sulphate test, to demonstrate differences in the rates of absorption of benzol by a number of individuals subjected to a given concentration. Yant has suggested that further experience with the method might very well show a correlation to exist between an individual's rate of absorption of a substance and his relative susceptibility to it. Generally speaking, however, this cannot be done because of limitations in our medical knowledge. However, a few guiding principles might be followed to advantage in the case of persons exposed to substances capable of causing serious liver damage.

1. Persons who have had a history of jaundice for any reason whatsoever should not work in exposure to known liver poisons. Similarly any worker who develops jaundice in the course of his work—whether or not it has resulted from such work—should be removed from further exposure immediately. Even after what appears to be complete chemical recovery he should be strongly advised never to work with these substances again. The risks are too great.

2. Persons who appear to be hypersensitive to these substances as evidenced by the development of characteristic dermatoses, for example, should not continue working with them. While the great majority of such persons never appear to develop any systemic disease, the fact that death from acute liver atrophy is sometimes preceded by a long-standing severe skin eruption would tend to suggest the desirability of regarding such an eruption as a danger signal. Our experience would seem to indicate that the prompt removal of such an individual from further ex-

posure may prove to be a life-saving measure in a given case.

3. It has been shown by Dr. Cecil Drinker, at Harvard, that rats which were apparently getting along perfectly well when exposed to certain chlorinated naphthalenes, if given a dose of carbon tetrachloride (too small to have any ill-effects on control animal), promptly died with evidences of acute yellow atrophy of the liver. On the basis of these observations, it would seem highly inadvisable for individuals who are taking drugs which are potentially toxic to the liver, or who are receiving injections of arsphenamine for the treatment of syphilis, to work with substances known to be liver poisons.

4. Similarly, persons working with these substances should not be given chloroform or avertin anesthesia if they must undergo a surgical operation. Vice versa, persons who have recently been given these anesthetics should not immediately go to work with substances which are toxic to the liver.

5. Pregnant women should never be exposed to liver poisons, since the liver, in pregnancy, is peculiarly sensitive to injury.

WHEN one considers the important question of prophylaxis, or the treatment of liver disease, one is impressed with how little is definitely known on the subject. However, it is generally believed that the liver is greatly supported in its ability to withstand injury by toxic substances, and that the effectiveness of regenerative processes materially increases when the glycogen reserves of the liver are high.

Minot has shown, moreover, that when calcium is administered, the ill-effects of liver poisons, such as carbon tetrachloride, appear to be definitely lessened. He explains this by the antagonistic action of calcium upon guanidin—an accumulation of which is produced by carbon tetrachloride absorption; and he believes the guanidin may be responsible for the clinical picture observed in these cases. Incidentally, experimental guanidin poisoning is relieved by calcium therapy.

For prophylaxis, therefore, to the best of our present knowledge, it would seem desirable for workers exposed to substances potentially capable of causing liver damage, to have a diet rich in carbohydrates and milk, supplemented during the day by a fair amount of candy or sugar in some other form. Similarly intravenous glucose and calcium seem to be used with good effect in the treatment of acute cases. The position of the diabetic worker in this situation is a matter for interesting speculation.

Some time ago Sato obtained an extract of the liver which he called "Yakreton." If administered properly, he believes his experiments have shown that this extract has the power to protect the liver of animals not only against toxic substances but against bacteria and a great variety of other injurious agents as well.

More recently Neale and Winter obtained a crystalline substance from the liver which, if injected into animals 24 hours in advance of exposure, definitely protected them against the characteristic liver damage produced by carbon tetrachloride and chloroform. They purified this substance and identified it as "sodium xanthine." Later they even synthesized it and got the same physiological response with the synthetic product.

Clinical data with the use of these extracts is not available as yet, and no conclusions can be reached as to what their efficiency in human beings might ultimately be. However, though still in their infancy, these experiments are very suggestive. Perhaps better methods than we have any notion of at the present time will be available in the near future for the protection of workers exposed to substances potentially injurious to the liver.

Silicosis

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DURING the past few years many states have enacted laws to award compensation to workmen who develop occupational diseases. One of the most important and widely discussed of these diseases is silicosis. It is important to the worker who contracts the disease, to the doctor who has to diagnose and treat the disease, to the employer who wishes to prevent it, and to the insurance carrier who, as a rule, is interested because he has to pay compensation for disablement or death. A year ago the State of Indiana passed an Occupational Disease Act, and those of us who are interested in industrial problems have been besieged with questions concerning silicosis by physicians, by employers of labor in the dusty trades, and by insurance companies.

The recent literature contains many very excellent articles on silicosis, but most of these articles go into the subject in such detail that it is difficult for the family physician and others interested in the subject to grasp the essential facts for their practical guidance.

This article is simply a brief compiled from the recent literature on silicosis.

Silicosis, a condition due to the inhalation of silica dust, is classed under pneumoconiosis, or the condition of "dusted lung." Other forms of pneumoconiosis are the following:

Anthracosis, the inhalation of coal dust by miners and those who handle coal;

Chalicosis, due to the inhalation of lime stone dust;

Siderosis, in workers in iron mines; and

Asbestosis, which may occur in those working in an asbestos dust-laden air.

Of all the forms of pneumoconiosis, silicosis and asbestosis are the only ones that constitute a definite form of lung pathology. All other mineral dusts are practically harmless, even when inhaled in large quantities and over long periods of time. The dusts of coal, calcium, and iron may become

deposited in the small lymphatic vessals of the lungs and also in the glands of the hilum. These dusts may show in the x-ray film as increased shadows about the hilum and also as an exaggeration of the normal linear markings of the lungs. However, such mineral deposits never cause enough scar tissue reaction to encroach upon the normal lung capacity[1]. In fact all forms of pneumoconiosis, with the exception of silicosis and asbestosis, should not be considered as true diseases. The dusts of coal, iron, and limestone, when inhaled, simply cause dirty lungs. A dirty skin is not necessarily diseased, but the dirt may lead to a skin irritation; so a dirty lung, while not actually diseased, may cause a moderate amount of bronchial irritation. However, this is true only for the pure mineral dusts or those containing 5% or less of silica. The dusts of hard coal and of several of the iron ores often contain a large percent of silica in which case they are capable of producing silicosis in proportion to their silica content.

The most concise definition of silicosis is given by Lanza[2] as follows: "Silicosis is a disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and by characteristic x-ray findings."

Etiology

THE cause of silicosis is one substance only, silica dust, and this must be inhaled into the lungs. Dusts composed of the silicates and other substances that may cause the various forms of pneumoconiosis never cause silicosis unless these dusts are mixed with silica dust.

The earth's crust contains an enormous amount of silica in many different physical forms, the most common forms used in industry being the following: Quartz, quartzite, sand, sandstone, flint, tripoli, granite, chert, and diatomaceous earth. Less common forms are rock crystal, amethyst, rose quartz, agate, jasper, onyx, opal, and other forms of semi-precious stones. Several ores, especially coal and iron, contain a variable amount of silica.

The chief industries and activities which may constitute a dust hazard from the inhalation of one or more of the above-named forms of silica are the following: Building highways, driving tunnels and working mines through silica-bearing areas and regions, using silica as an abrasive in the form of sandstone, as an abrasive powder and in sand blasting, in the manufacture of fire brick, glass, pottery and glazed ware, as a flux in smelting ores and making alloys, as a filler, in the manufacture of certain substances, for surfacing roads, as a heat insulator, and in the manufacture of articles from granite and other quartz-bearing stones.

While the above-named industries and operations bring their workers into contact with silica dust, it does not follow that they will all develop silicosis. In order to develop this disease the silica dust inhaled must possess certain definite requirements.

First, the size of the dust particle is most important. In order for a particle of silica to produce its harmful affect it must first enter the lung alveolus, and in order to be able to enter this small sac it must be 10 microns or less in diameter. All larger particles are swept out by the cilia of the bronchi and bronchioles. In most cases of silicosis the dust particles range between 3 and 4 microns.

Next, in order to produce silicosis there must be a certain amount of silica dust inhaled. In other words, the concentration of the dust in the air must reach a certain amount. This concentration is expressed by the number of dust particles in a cubic foot of air. It is generally considered that a silica dust atmosphere containing no more than 5 million particles per cubic foot is perfectly safe[3]. Concentrations above this are dangerous in proportion to the degree of concentration. If the dust particles contain some substance other than silica, the number of particles per cubic foot can be raised with safety. For example, hard coal often contains from 30% to 35% silica, so that it would require around 15 million particles of such coal dust per cubic foot to reach the upper limit of safety.

The next factor in producing silicosis is the length of time the worker is exposed to the dust. It has been estimated that it takes an exposure of two to three years to develop the disease. This is probably the average minimum time. It is stated that: "Usually it takes seven years or more of exposure to silica dust for a worker to contract the disease. It is said that a few cases have been known to develop it in as short a period as one and one-half years, but these were under extreme and unusual conditions."[4]

As far as we know there are few factors that predispose an individual to the development of silicosis, all workers under similar conditions being equally susceptible. It has been pointed out "that perhaps not more than 50% of the dust in the atmosphere at the breathing zone enters the lung because of protecting mechanisms. These consist of coarse hairs, which guard the nostrils; the sticky mucus secreted by the cells lining the nose, throat, trachea and bronchi, and the cilia" of the bronchial tubes. It has also been observed that mouth breathers and those who have nasal disorders that reduce the filtering function of these passages show a less resistance to the effect of inhaled silica dust.

In order, therefore, for a person to contract silicosis, he must be exposed to a certain concentration of silica dust, the grains of which are 10 microns or less in diameter, this exposure to continue for a period of two or more years.

Pathology of Silicosis

THE action of silica upon living tissues lends itself to a complete study, as "all species of mammals, birds, amphibians and fish that have thus far been tested react to silica in a specific manner,"[5] so that our knowledge of the pathology of silicosis is not confined to human postmortem findings alone. It has been definitely proved that silica

within the lung alveoli acts purely on account of its chemical nature, producing a diffuse nodular fibrosis rather evenly distributed throughout both lungs. The physical properties of the silica particles play no part in the fibrotic changes, as "hard, sharp particles of diamond, aluminum oxide and carborundum cause no fibrosis."[6] It has also been demonstrated that silica in solution or in a colloidal or non-crystalline form, when injected into the tissues, leads to a proliferation of fibroblastic cells. In other words, silica acts upon the lungs because of its chemical nature rather than as a physical agent.

Silicosis is a progressive disease, and the amount of fibrosis in the affected lungs is about in proportion to the length of time the worker has been exposed to silica dust. Variations in this progression will depend upon the variations in the concentration of the dust particles, and also upon the percent of silica in the particles. It has also been definitely shown, both in man and in animals, that the complete removal from the silica exposure does not stop the progression of the disease at once, but that the fibrotic changes continue to develop for another one to two years, probably due to the continued action of the silica that has been stored in the phagocytic cells and lymphatics.[7]

There is considerable evidence that several of the compound dusts containing silica produce less damage than would occur if the same amount of pure silica were inhaled. In other words, the substance mixed with the silica exerts a protective action inhibiting to a certain degree the action of the silica upon the tissues. It has been noted that granite cutters develop silicosis only after very long exposure, that workers in calcined gypsum (plaster of Paris) seldom develop silicosis even when they have been subjected to long periods of work in an atmosphere of silica dust. To a less degree the same is true of the dust of iron ore when naturally mixed with silica.[8]

As mentioned above, the chief action of silica upon the lung tissue is to produce a diffuse nodular fibrosis. At first these nodules are so small that they can only be seen with the microscope. As the disease progresses, these nodules grow larger, and become visible to the naked eye on gross section of the lung. They can also be seen as small round shadows on the x-ray film. With further progression, these nodules become still larger, and finally coalesce. With the progression of the nodular fibrosis there is a gradual encroachment upon the normal air space of the lungs, but even in extreme cases it is rare to find the total reduction in air space to exceed the residual air capacity of the lungs. With the obliteration of air cells of the lungs there is a certain degree of compensatory emphysema.

The fibrotic nodules may extend to the surface of the visceral pleura, but uncomplicated silicosis never causes a pleurisy or pleural adhesions.

Considerable work has been done to determine whether or not silicosis occurs in other organs or tissues in those exposed to silica dust. In experimental animals it has been shown that silica can cause fibrosis in most organs and tissues, but in man, even when suffering from a most pronounced grade of silicosis of the lungs, the silicotic fibrosis in other organs is so slight as to be of no pathological or clinical significance.

Silicosis is not accompanied by fever or any form of toxic disturbance.

Symptoms

THE symptoms of uncomplicated silicosis are directly proportional to the pathological changes in the lungs, that is, the reduction of air space by the fibrotic nodulation. The disease may be well advanced before the patient becomes aware of any discomfort. As the reduction in lung air space continues he may notice shortness of breath on exertion. This is the chief symptom of uncomplicated silicosis, and it seldom reaches a degree sufficient to disable the patient unless he is required to put forth an unusual effort. The margin of safety of the lung capacity is a large one, as an individual can carry on with only one-fourth of his normal capacity. "He has merely reduced his reserve capacity and only manifests shortness of breath when sudden violent exercise creates a sudden demand for a large amount of oxygen."[9]

Dizziness, general weakness, and headache have been mentioned as symptoms of silicosis, but in most cases these symptoms are directly associated with the dyspnea.

Due to the fibrotic changes the lungs become less elastic and the normal chest expansion is reduced.

The physical signs found in uncomplicated silicotic lungs are of little importance. When the silicosis is accompanied by one of the other forms of pneumoconiosis the signs of chronic bronchitis may be present. When complicated by tuberculosis, however, the physical signs become most important.

X-Ray Changes in Silicosis

IN ORDER to study and diagnose silicosis by means of the x-ray, one must be quite familiar with the normal lung markings and also the markings due to the non-silica dust reactions. Everyone living in a dusty or smoky city shows on the x-ray films of his lungs an exaggeration of the normal lung markings. Those working in the non-silica dusty trades show an even greater exaggeration of these markings, the so-called linear markings or linear bands that radiate from each hilus along the course of the blood vessels, bronchi, lymphatics and interlobular septa, together with an increased density of the tissues at the roots of the lungs. Similar markings may occur in persons suffering from chronic pulmonary infections, chronic bronchitis, and in circulatory disturbances in which there is a chronic passive congestion of the lungs. These linear markings also become more marked with advancing age.

The x-ray films of a silicotic person who is free of complications or infection may be classed in one of three stages:

1. Stage of perivascular, peribronchial, and lymph node proliferation, together with an irregular exaggeration of the linear markings.
2. Stage of diffuse fibrotic nodulation.

3. Stage of coalescence of the fibrotic nodules, with the formation of conglomerate masses.[10]

Stage No. 1 is the pre-nodular stage and shows itself on the x-ray films only as an exaggeration of the linear markings. A diagnosis in this stage can seldom be made either from the x-ray or the clinical findings, but the presence of these markings in an individual working in an atmosphere of silica dust should call for repeated x-ray films made every four to six months.

Stage No. 2 is the stage of discrete nodulation. "Gross examination of such lungs shows sharply defined, hard fibrous nodules, not over 4 mm., and usually 2 to 3 mm. in diameter, uniformly distributed throughout both lungs. A roentgenogram shows these clear cut nodules uniformly distributed throughout both lungs with or without associated clear zones of emphysema in the outer and lower portions. The shadow of the mediastinum is wide if the contained tracheobronchial lymph nodes are enlarged."[11]

Stage No. 3 appears when the fibrotic nodulation progresses to the point that the nodules coalesce to form conglomerate nodular masses.

X-ray films of pure uncomplicated silicosis in the second and third stages are, as a rule, fairly easy to diagnose, but when the dust is composed of silica plus another mineral such as coal or iron the x-ray picture will be a combination of the nodulation of silicosis plus the linear markings of the anthracosis or siderosis. A silica content of an ore dust no greater than 5% is harmless and cannot be demonstrated on the x-ray film. A silica content ranging between 5% and 25% shows nothing but an exaggeration of the linear markings, that is, a first stage. With a silica content of 25% or more a definite silicosis may occur, and the x-ray films will show a combination of the nodular fibrosis of silicosis together with the linear markings of the other mineral. "The picture produced by a mixture of silica and other substances is characterized by its variable nature. Depending upon the amount of silica entering the lung, one may find anything from an almost pure linear or perilymphatic type of reaction, ordinarily produced by non-silicious dusts to nodular lesions with or without the central core of more typical silicotic fibrosis. Such pictures are encountered in the anthroco-silicosis of hard coal miners or the siderosilicosis of hematite miners."[12,13]

No matter whether the silica is pure or mixed with some other mineral, the silicotic evidence on the x-ray film is always in the form of fibrotic nodulation. This nodulation can only be confused with miliary tuberculosis and metastatic malignancy of the lungs, but when these conditions are present the clinical evidence will make clear the differentiation. Certain forms of fungus infection of the lungs may be confusing. In this case there is an exaggeration of the linear markings plus diffuse shadows in both lungs, but these shadows are never definitely nodular.

So far we have considered silicosis uncomplicated by infection. Many men so affected are unaware of their silicosis and continue in their dusty trades for periods of 30 or more years. Such cases are found because of the routine x-ray examination of the lungs now required by many employers of labor in the various dusty trades. A few of these men complain of shortness of breath on exertion due to the reduced air capacity of the lungs, while others show a varying degree of associated chronic bronchitis. Both of these conditions or complications in a certain number of silicotic persons may finally reach such a degree as to cause a partial or total disablement.

Silicosis and Tuberculosis

FROM the foregoing we can conclude that simple silicosis of itself does not constitute a particularly serious industrial problem, but in a great many instances it may develop into a very serious problem owing to the fact that persons who have developed silicosis are very likely to develop tuberculosis of the lungs.

Gardner stated: "At least 75% of those human beings who develop silicosis die of tuberculosis which may make its appearance at any stage of the disease."[14] This percent is high compared with recent figures; nevertheless, we must keep in mind that pulmonary tuberculosis is a serious and common complication of silicosis.

Pathology of Silico-Tuberculosis

LABORATORY and clinical studies have demonstrated that the presence of silica aids in the growth of the tubercle bacilli. "Prince, of Toronto, demonstrated that the addition of soluble sodium silicate to artificial culture media accelerated the growth of the tubercle bacilli."[15] "In the animal body, Kittle, the Saranac Laboratory group, and others, have demonstrated that tubercle bacilli begin to grow quite rapidly in tissues that are reacting vigorously to injected or inhaled silica."[16]

A person with silicosis may develop tuberculosis in one of three ways:

1. He may have a quiescent or clinically healed focus of old tuberculosis in one or both lungs. With the establishment of the silicosis the old infection is reactivated. This is probably the most common method of developing silico-tuberculosis.

2. A person may be suffering from an active pulmonary tuberculosis which has not reached a disabling stage. He obtains employment in a silica dust trade. In such a case the silicosis develops after the tuberculosis, but the presence of the silicosis rapidly aggravates the infection.

3. A person may develop silicosis and remain free from symptoms for a few to many years. He then contracts tuberculosis from some outside source, either from a fellow worker or from a contagious source at his home. In such a case the tuberculosis will progress more rapidly than when implanted upon a non-silicotic lung.

Symptoms and Findings in Silico-Tuberculosis

THE symptoms and physical signs of silico-tuberculosis differ little from those found in the various forms of non-silicotic pulmonary tuberculosis. One point of difference is that in silico-tuberculosis the bases of the lungs are more fre-

quently involved than the apices. Again the demonstration of the tubercle bacilli in the sputum is often difficult in silico-tuberculosis.

X-Rays of Silico-Tuberculosis

THE x-ray films of a patient suffering from silico-tuberculosis show the shadows of silicosis plus those of tuberculosis. The interpretation of such films may at times be most difficult. In many films showing silicosis plus the shadows of old healed tuberculosis it may be impossible to determine by x-ray if the latter has become active. In these patients the clinical features of the case are most important. Again, the presence of tuberculosis in a silicotic lung of the third stage in which the film shows many conglomerate masses may be very difficult to determine.

From our experience we feel that the diagnosis of uncomplicated silicosis can be made by the average physician after studying several of these films, but we also feel that if there is any question of the presence of silico-tuberculosis the films should be studied by a trained roentgenologist.

Practical Considerations

THE National Silicosis Conference of 1937[17] gives the following figures:

It is estimated that in the United States there are:

49,000,000	workers.
1,000,000	are exposed to some form of silica hazard. Of these
500,000	are exposed to a serious hazard.
110,000	have silicosis of some degree. Out of this number
5,000	suffer some form of disablement. Of this group the majority will have a silico-tuberculosis. The minority of this group will be only partially disabled on account of chronic bronchitis or of reduced lung air capacity.
105,000	have silicosis but no disablement.

Regarding the above figures the Conference divides them into four groups and makes the following observations:

GROUP 1. This contains that part of the 5,000 who have silico-tuberculosis. These men are to be considered totally disabled until their tuberculosis is either arrested or healed.

GROUP 2. "The remainder of the 5,000 workers who have silicosis and disability in some degree, but no tuberculosis, should in the majority of cases continue to work. While a few persons may still be of the opinion that such workers are 'through' and doomed to an early death, every study conducted thus far confirms the fact that the great majority of these men will not necessarily progress from bad to worse. Perhaps it may be wise to transfer some workers to other employment, but many may be continued at their regular occupations if known methods are applied to control the dust and if special care is employed to prevent the development of tuberculosis, pneumonia and other lung complications."

GROUP 3. "To the 105,000 workers who have silicosis, but no disability and no tuberculosis, the problem is not particularly serious. It is generally agreed that, if the dust is properly controlled at all times, such workers would be as well off in continuing at their present jobs as if they were discharged or transferred to other employment. Therefore, such men should be permitted to continue work at their regular occupations, but known methods of controlling dust should be applied and care should be used to prevent the development of tuberculosis, pneumonia, and other lung complications."

GROUP 4. For the benefit of the approximately 900,000 workers who are exposed to silica dust but who do not have silicosis, all emphasis again should be placed upon the application of known methods of controlling dust."

In this Conference it is also stated: "From a purely statistical point of view, therefore, the problem of silicosis is not as serious or as general as some other industrial problems, such as lead poisoning and industrial accidents."

Many physicians will be asked by the employers of labor in the various dusty trades as to the best way of handling their silicosis problems. These employers should be advised, in brief, as follows:

The dusty air surrounding the workmen should be investigated by a safety engineer to determine the number of dust particles 10 microns or less in diameter, per cubic foot of air. The silica content of the dust should be determined chemically. These facts will determine whether or not there really is a silica hazard, and to what degree it is present.

If a silica hazard is present the next step is an engineering problem, to reduce the hazard by "good-housekeeping" shop practice, by proper ventilation, and, where the dust cannot be eliminated, to furnish respirators to the workmen. (However, it has often been found difficult to persuade the men to wear these.) If the silica hazard cannot be eliminated, the workmen should be rotated in their jobs so that for two to three days a week they can be removed from the dusty atmosphere.

The physician's duty when a silica hazard is present is to examine all of the exposed workers. This should consist of a general physical examination together with a flat film of the lungs, taken under standard conditions. If the findings are unusual or difficult to interpret in the flat films a stereo set of films should be made. From these examinations the physician should be able to place the workmen in one of the four groups as mentioned above.

GROUP 1. Those with definite silico-tuberculosis. These men are totally disabled and should be placed where they can obtain proper treatment.

GROUP 2. Those with silicosis who are free from tuberculosis, but who are partially disabled because of chronic bronchitis or from a reduced air capacity of the lungs. These men should be removed from the silica dust atmosphere and given work suitable for their general physical condition.

GROUP 3. Those who have silicosis, but who are free from symptoms. These men are not disabled, but they should be removed from the influence of silica dust and allowed to do other types of work.

GROUP 4. These are the men who have been working in a silica dust atmosphere, but are free from silicosis. These should continue to work, but alternating their jobs so that for two to three days

a week they can be in a silica dust free atmosphere.

In all forms of work in which there is a silicosis hazard all of the men should have a flat film of the lungs made at least once a year. There will be a certain number of cases in which it will be impossible to determine definitely whether or not there is a beginning silico-tuberculosis. In such cases a physical examination plus x-ray films should be made every three to six months.

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Management of Compression Fractures of the Dorsi-Lumbar Spine

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UNTIL the last decade the treatment of compression fractures of the dorsi-lumbar region was neglected to a status whereby a patient was put to bed to remain on his back for a considerable period of time. The fear existed that if any interference was done, barring the necessity where a laminectomy was indicated for the relief of nerve symptoms, the condition would be made worse.

During back rest the patient had difficulty in turning on account of pain, so that pneumonia was not an infrequent complication. Despite prolonged bed rest on a fracture board, or a Bradford frame, there was not adequate correction of the deformity, so that when these patients recovered they were left with "lame" backs due to faulty vertebral alignment. Backaches were of such nature that the ability to resume work that necessitated activity of the back was definitely impeded.

Wih modern methods of adequate treatment a patient who has a compression fracture without paralysis should get a result which will permit intense activity of the back.

Given a history of a fall from a height, with pain and stiffness of the back, an examination is made to determine areas of definite bony tenderness.

This point is stressed here, as often one is misled in the localization of a spinal fracture due to radicular pain away from the site of injury. The localized tender area at times associated with a prominence of the spinous process is essential for proper localization of the x-rays which are taken. The views are the antero-posterior and the lateral. The lateral is the most important to show wedging or compression of the body. In examining x-rays, one is struck by the various phenomena of compressions. In some cases the impaction is such that the line of fracture is difficult to visualize. The site of compression is usually the upper surface of the body involved.

The most frequent fracture sites are the lower dorsal and the upper lumbar vertebra, with the twelfth dorsal and the first lumbar chiefly involved.

If the patient can be easily turned, oblique views are taken to note any fracture of the lamina or the articulations.

In 1929, Davis[1] showed brilliant results with manipulation followed by plaster fixation. He proved by clinical results and x-rays that anatomic restoration and a useful back are possible. A number of workers followed, demonstrating excellent results obtained by different methods. All have agreed that hyperextension is the necessary goal to disimpact the compression of the anterior portion of the vertebra that results, due to the usual mechanism of the injury which is a jack-knifing of the spine.

Wm. Rogers[2] constructed a frame for producing gradual hyperextension. The apparatus is ingenious and has given excellent results to those workers who have used this procedure.

Watson Jones[3] simplified the situation by placing the patient prone between two tables, separated so as to allow the body to sag, thereby producing the desired hyperextension. He obtained results without the use of unusual apparatus by this simple method that requires no anesthesia except preliminary morphine.

Bohler commended Watson Jones' technique, except varied in that he gives the patient perispinal nerve blocks with novocain before placing in position.

Conwell[4] has modified the convex frame of Herzmark, and has recorded excellent results with this form of hyperextension.

Ryerson[5] places an automobile jack modified with a supporting plate at the site of fracture and produces hyperextension.

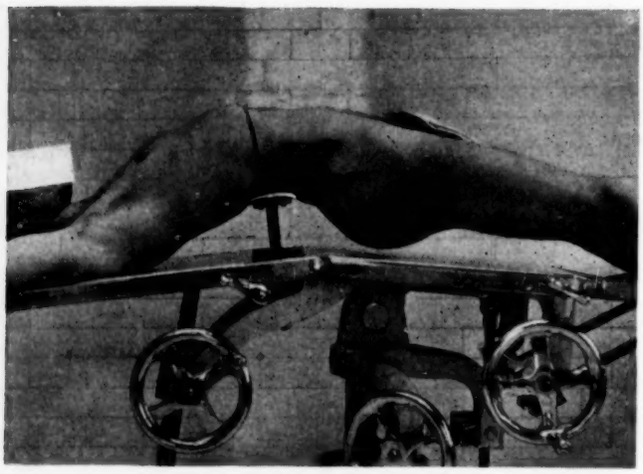
All of the methods have given excellent results, not only to the originators, but in reviewing the literature there is also found adequate testimony of the efficiency of these methods by others.

IN THE past we have had occasion to use some of these procedures. As with many others, we do not as yet feel confident to use forceful manipulation as advocated by Davis. The methods of Rogers and Conwell require special apparatus with which the average institution is not equipped. Placing the patient between tables as outlined by Watson Jones is simple and is advocated. We have found hyperextension by this means at times inadequate, owing to marked spasm of the back muscles and the protective spasm of the abdominal muscles. The automobile jack, although readily available, is frequently difficult to balance against the desired area to be compressed, due to marked local pressure.

Within the last two years we have used a simple method for producing hyperextersion in such a way that a force of direct compression is produced on the posterior aspect of the spine at the site of the fracture with adequate counter pressure of the upper portion of the body and the lower extremities.

The armamentarium necessary is available in every hospital. The effect is obtained by jacking an operating table on which a kidney plate is placed at the site of the fracture.

METHOD: Preliminary morphine is administered. Before commencing the procedure about 30 cc. of 1% solution of novocain are injected peri-spinally at the site of the fracture. A stockinette is applied. Circumferential marking is made across the chest or abdomen at the site of fracture, using a colored solution, as mercurochrome. The patient is placed on the table in a supine position with the fracture site at the middle of the kidney plate. The wheel controlling the kidney plate elevation is turned gradually. At the same time, the leaves of the table are dropped until an exaggerated degree of hyperextension is obtained. At this time, padding is applied and a heavy anterior lateral plaster shell is molded that extends from the upper sternum to the groin. This shell is reinforced by temporary flannel bandage or, if there is sufficient space to work under the table, the back is encircled with plaster bandages. After the shell hardens the patient is turned prone and placed between two tables and the back portion of the cast is completed.



A simple method—using an operating table with a kidney attachment

The contra-indication to the use of hyperextension is where there is gradual paralysis. Should there be evidence of associate fracture of the lamina with the body there should be caution as to forceful hyperextension, due to possible compression of the posterior neural arch into the cord.

When should reduction be done? There are many advocates who suggest immediate reduction. From experience, it is deemed advisable to wait several days to watch for possible paralysis of the lower extremities. Abdominal paresis sometimes develops the first few days, which results in marked abdominal distention. With a cast applied, even after a window is cut in the cast, the pressure is often unbearable. It is possible to disimpact a compressed vertebra without difficulty within the first 10 days after an injury.

The cast is maintained for about eight weeks, and it is often advisable to maintain bed rest for about 12 weeks in all. During cast and bed confinement free movement is permitted. It is essential that the patient be made to do simple exercises such as leg raising and extension of the back muscles by simple means to maintain muscular tone. Following, a well-fitting brace is applied which is worn for about six months.

SUMMARY: A brief resumé is given of some of the accepted methods for reduction of dorsi-lumbar compression fractures.

A simple method available in any hospital using an operating table with a kidney attachment is presented.

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American Railway Surgeons

THE AMERICAN ASSOCIATION OF RAILWAY SURGEONS will hold its Forty-Ninth Annual Meeting at the Palmer House, in Chicago, on September 19, 20, and 21, 1938.

DR. HARVEY BARTLE, Chief Surgeon of the Pennsylvania Railroad is President of the Association; DR. T. L. HANSEN, Chief Surgeon of the Rock Island Lines, is Treasurer; and DR. D. B. MOSS, Chief Medical Officer of the Burlington Lines, is Secretary.

Scientific and commercial exhibits will be an attractive feature of this convention. The program of the sessions will be announced in an early issue of this magazine.

Acute Lead Poisoning: Industrial

—II. Laboratory Studies in 50 Cases—

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IN A preceding communication,[1] we presented the results of a study of the history and physical findings in a group of 50 men, who had acute lead poisoning. In this publication we are reporting the results of the laboratory studies in this same group of 50 men.

These studies included:

I. Blood Examination:
   (a) Red blood cells and hemoglobin.
   (b) White blood cells.
   (c) Differential count.
   (d) Platelets.
   (e) Basophilia.
   (f) Blood chemistry.
II. Renal function.
III. Liver function.
IV. Gastric function.
V. Lead in biological materials, (stool, whole blood, and urine).

These patients were not exposed to any single industrial hazard. They included riveters, painters, water-tank cleaners, and lead smelters, in the following order of frequency:

CHART I
OCCUPATIONAL DISTRIBUTION OF 50 PATIENTS

Occupation	No. of Cases
Riveting in enclosure	35
Painting	9
Water tank cleaning	4
Lead smelting	2

The riveters[2] were engaged in the construction of a tower of a bridge. They inhaled lead fumes and lead dust within an enclosed chamber, when the hot rivets melted the red lead covering the steel.

At no time previously, in the many years of their occupation, did they have any symptoms or signs of lead poisoning, by reason of the fact that their work was done in the open. All 50 patients were admitted to the hospital for treatment. In the entire group, whenever possible, biological specimens were obtained and studies of the renal, hepatic and gastric function were done, prior to any attempt to influence the lead metabolism.[3]

I. Blood Examination

RED BLOOD CELLS AND HEMOGLOBIN:

The hemoglobin was estimated by the Sahli method. It ranged between 70% and 88%, with an average for 50 patients of 75%. The red blood cells ranged from 3.0 m. to 4.2 m., with an average of 3.6 m. for all patients. Aub[4] reported that the irritating effect of lead on bone-marrow ultimately produced a definite change, resulting in a decreased output of normal red cells, a release of immature cells and a relative increase in the number of leukocytes. Hamilton[5] stated that "stippled cells are really degenerated red blood cells." In this author's opinion, changes in the blood cells constituted the earliest sign of lead intoxication. The development of symptoms in the gastro-intestinal tract or nervous system may precede the blood changes where there is a sudden and large increase in exposure to the lead hazard or where there is a complicating illness. If the exposure is comparatively regular and quantitatively constant, the first sign is likely to be a drop in the hemoglobin content of the blood. This is frequently accompanied by an increase in stippled red blood cells and a diminution of the red blood cells.

(B) WHITE BLOOD CELLS:

In this group of 50 patients, the total white blood cell count ranged from 6,000 to 18,000. Polyneucleosis was noted in a group of seven individuals whose oral hygiene was poor. Excluding this group of patients, the count was well within normal limits in the remaining 43 patients. Bell, Williams and Cunningham,[6] in studying a group of patients who had received intravenous injections of colloidal solutions of lead for the treatment of carcinoma, reported that the initial leukocytosis, which reached its height within 48 hours after the injection was given, ranged as high as 20,000. However, whether this was due, as the authors noted, to the action of the lead on the reticulo-endothelial system or whether it was due to the autolytic process and absorption of the foreign protein often associated with malignant diseases, could not be determined.

(C) DIFFERENTIAL COUNT:

A uniform smear was made and stained with the Wright stain. The differential study was then carried out under oil immersion. Twenty-five patients (50%) had a lymphocyte count of over 25%. The highest lymphocyte count was 46% and the lowest was 12%. The average for the entire group was 29%. The large mononuclear cell count in nine (18%) of this group was 3%. The highest mononuclear count was 5%. Fifteen (30%) of the patients had an eosinophilia of 3% or more. The highest eosinophile count was 7%. We were unable to elicit, from any of these 15 patients, a history of any allergic state, which might account for the eosinophilia. We raise the question here, whether or not the ingestion or inhalation of lead can produce an eosinophilic response in the normal individual, similar to that seen in atopic individuals. Mayers,[7] found that 62% of 381 cases of lead poisoning which she studied, had a lymphocyte count of over 35%. The large mononuclear cells in this group were increased in 30% of the cases studied. Legge and Goadby,[8] consider a mononucleosis characteristics of lead poisoning. Shie,[9] found an increase in the large mononuclear cells in his cases.

(D) PLATELETS:

In a study of 42 individuals with "moderately severe plumbism" Jones,[10] noted an average platelet count of 130,000 per cubic millimeter. In 70% of our cases the platelet count was below 200,000 per cubic millimeter. In one of our cases we noted a platelet count below 100,000. In spite of the marked reduction in the number of platelets, this patient had no evidence of hemorrhagic tendencies. The bleeding time in this group was within one to three minutes and coagulation time within four to eight minutes. There was no relationship between the platelet count and the coagulation time.

(E) PUNCTATE BASOPHILIA:

Stippling of the red blood cells or punctate basophilia has long been recognized. As early as 1885, Ehrlich,[11] described this condition as the presence of basophilic granules in certain red cells. Basophilic stippling in association with lead poisoning, however, was first pointed out by Behrend,[12] in 1889. The attention of many investigators was immediately focused on the study of stippling, its causation, its relationship, and, more especially, its significance.

Because of the completeness of the recently published work of Pappenheim[13] and Naegli,[14] a detailed account of the results obtained, and the theories evolved, concerning stippling of the red blood cells, is unnecessary here. Punctate basophilia is present in practically all workers exposed to the lead hazard. The larger the granules the greater the significance which should be attached to stippling as denoting excessive absorption of lead. Sellers,[15] has remarked on the different types of granules found in red blood cells and has attached significance to the size of the granules in relation to the severity of the intoxication.

Key,[16] noted that polychromasia, punctate basophilic stippling and reticulation were manifestations of but one phenomenon, namely, the presence of basophilic substance in the protoplasm of the red blood cell. Hawes,[17] believing that polychromasia and basophilic stippling represented but different phases of the same process, made no distinction between cells exhibiting both phenomena and reported them as one. Jones,[18] was of the opinion that basophilic substance could be demonstrated as a form of polychromatophilia, punctate basophilia or reticular designs, depending upon the staining method used. The presence of stippling in the blood smear, must be regarded with caution.

The presence of stippled cells in a blood smear was indicative of lead absorption, according to Marks,[19] if primary anemia, the leukemias, malaria and benzol poisoning could be excluded. It is not possible to estimate the amount of absorption of lead nor the duration of exposure to the lead hazard from a determination of the number of the stippled cells. Furthermore, it is not possible to estimate the severity of the intoxication, nor judge the progress of the case of lead poisoning from a study of a single blood smear.

The method of Lane,[20] was used in our studies to report the number of stippled cells. A uniform smear was made and stained with the Wright stain. Study for stippling was then done under oil immersion. It was noted (as described by Lane) that there were approximately 200 cells per field. One hundred fields were searched for the presence of stippled cells. Stippling was reported in terms of the number of cells seen per one million red blood cells.

The stippled cell count in some of our cases was as high as 1500 stippled cells per one million red blood cells (15 per 50 oil immersion fields). Belknap,[21] attached great significance to repeated study of blood smears for stippling. He believed that the presence of five or 10 stippled cells per 50 fields (500-1000 per one million red blood cells) does not necessarily indicate anything more than lead absorption. McCord[22] and his co-workers noted that the qualitative demonstration of stippled cells proved of no value in the diagnosis of clinical lead poisoning.

In our group basophilic stippling was present on the control smear in 20 (40%) of the patients. An average of 10 stippled cells per 50 oil immersion fields was noted. Our studies did not indicate any relationship between the size of the granules and the severity of the clinical symptoms.

(F) BLOOD CHEMISTRY:

Blood was drawn routinely on admission for chemical study.

CHART II

BLOOD CHEMISTRY STUDIES IN ACUTE LEAD POISONING

	No. of Cases	Minimum per 100 cc. of Blood	Maximum per 100 cc. of Blood
Sugar	43	86 mgm. per 100 cc.	114
Urea	40	10.9.	33.8*
Creatinine	39	1.1	2.3
Uric acid	31	2.1	8.6*
CO_2 combining power	10	48.7 Vol. %	61.3 Vol. %
Calcium	42	8.6	13.8*
Phosphorus	42	1.1	6.0

* One patient had a retention of nitrogen.

A review of the second chart indicates that the values obtained for, sugar, urea, creatinine, uric acid, carbon dioxide combining power, calcium, and phosphorus were within normal limits. One patient had a retention of nitrogen. This was a white male adult, 45 years of age, who had a blood urea of 33.8 milligrams, and a creatinine of 2.3 milligrams. Studies indicated that we were dealing with a case of acute lead poisoning complicating chronic glomerulonephritis. This worker had been exposed to the lead hazard for 15 years, and stated that he had never been ill prior to the onset of the acute abdominal episode, so frequently seen in lead poisoning. The patient denied having had any of the infectious disease of childhood. On the very first examination nitrogen retention, low hemoglobin and red blood cell count, and albumin and casts in the urine were noted. There was no history of any of the childhood exanthematous diseases, nor was there any history of recurrent sore throats.

Glomerulonephritis is a slowly developing disease which is often insidious in its onset. The disease progresses unnoticed up to the stage when the renal damage is so great that the kidney can no longer carry on its function adequately. Such a sequence of events is not uncommon in the non-industrial population. Not infrequently a diagnosis of chronic glomerulonephritis is made and nitrogen retention is noted. Yet as far as we can ascertain, a clear cut history of an etiologic factor is lacking. It is often difficult to set the time of onset of this disease with any reasonable degree of assurance. If this is true in the non-industrial population are we justified in attributing the uremia, which ultimately cost this patient his life, to lead which he absorbed as a result of his occupational exposure?

II. Renal Function

URINALYSES were done routinely on all of our 50 patients. These examinations included specific gravity and reaction. Albumin was tested for by means of boiling, plus the addition of acetic acid. Benedict's solution was used to test for sugar. Microscopic examination, for casts in the urine, served to indicate renal damage. Conversely, the absence of albumin and casts should, therefore, indicate fairly good renal function. On repeated examinations, the urine of all but the one patient mentioned above remained negative for albumin and casts. None of our patients had diabetes. Single specimens of urine showed a wide range of concentration. Further studies to determine the status of kidney function were carried out.

Chart III
Renal Function Tests in Acute Lead Poisoning

Procedure	No. of Cases	Result
Urinalyses*	49	No albumin, sugar, or casts.
Dilution and concentration test	10	Specific gravity varied from 1004-1025. Normal day-night ratio.
Urea clearance test	8	35-50 cc. of blood cleared of urea per minute.
Phenolsulphonthalein test	5	Normal amount (60 to 80%) of dye excreted in 2 hrs. and 10 min.

* One patient had glomerulonephritis.

The dilution and concentration tests were done to determine kidney function with a measured amount of fluid over a period of 24 hours. Specimens were collected at hourly intervals from 7:00 A.M. to 1:00 P.M. and then every two hours until 9:00 P.M. The night specimen was collected from 9:00 P.M. until 7:00 A.M. the following morning. The results of 10 such examinations indicated good concentrating power, a wide range of specific gravity, and a normal day-night ratio. Urea clearance tests were done on eight patients. Particular attention was paid to the time of collection of the specimens for study. The Addis ratio indicated that from 35 to 50 cc. of blood were cleared of urea per minute. Five patients were given 1 cc. of phenolsulphonthalein, intramuscularly about 10 minutes after they had taken a large glass of water. The bladder was emptied at the time of injection. Specimens were collected 1 hour and 10 minutes, and again, 2 hours and 10 minutes after the injection of the dye. Between 60 and 80% of the dye was recovered by the second hour. From the foregoing, one may conclude that the renal function of those patients who were studied remained well within normal limits.

III. Liver Function

AUB[23] and his colleagues have demonstrated experimentally the fate of lead which gains entrance to the body by way of the gastro-intestinal tract. Most of the lead passes through the alimentary canal unabsorbed and may be recovered in the feces. Carlson and Woelfel[24] have shown that even the most insoluble of inorganic compounds of lead, can be dissolved to some extent by the gastric juices. The greater portion of the absorbed lead is taken up by the liver and re-excreted into the intestinal tract via the bile. Lead is known to be stored in the liver in acute lead poisoning.

Chart IV
Tests of Liver Function

Procedure	No. of Cases	Result
Bromsulphalein test	5	Less than 15% of dye retained 1 hour after injection.
Glucose tolerance test	4	Normal curve, peak reached within 1 hour after ingestion of 100 grams of glucose and a return to normal within 2 hours. No sugar recovered from urine.
Galactose tolerance test	4	Less than 6.0 grams excreted in 4 hours.

In order to determine whether the liver was damaged by the lead which passed through it, the galactose tolerance, glucose tolerance and bromsulphalein excretion tests were employed as tests of liver function. The glucose tolerance test[25] was done on four patients. After fasting, blood specimen was obtained and each patient was given 100 grams of dextrose dissolved in two glasses full of tea, to which lemon juice had been added for flavoring. Specimens of blood were taken after half an hour and at the end of the first, second and third hours. Specimens of urine were obtained with each specimen of blood. The sugar content of each specimen was determined and curves were plotted for each patient. Each specimen of urine was tested with Fehling's solution to determine whether or not sugar was present. The peaks of the curves were reached within one hour, and the blood sugar level returned to normal within two hours in each instance. Sugar was not recovered from any of the specimens of urine.

The bromsulphalein test,[26] was done on six pa-

tients. Each patient was given one cubic centimeter of a 5% solution of the dye for each 10 kilograms of body weight, intravenously. At the end of half an hour, and again at the end of one hour, 5 cc. of blood were obtained from the other arm. Dry needles and dry test-tubes were employed to prevent hemolysis. The blood was permitted to clot and the serum was centrifuged. The amount of dye excreted was determined by colorimetric comparison with standard solutions. In our case less than 15% of the dye was retained at the end of one hour.

The galactose tolerance test[27] was done on four patients. Forty grams of galactose were dissolved in two glassfuls of water and given to the patient on a fasting stomach. Specimens of urine were collected hourly for five hours and were tested both qualitatively and quantitatively with Benedict's solution. We found that less than 6.0 grams of galactose were excreted in five hours.

IV. Gastric Function

FRACTIONAL gastric analysis was done on 20 patients. Following extraction of the fasting specimen, the patients were given two crackers and a tumbler of water. At 15-minute intervals, extractions were done until six samples were obtained. Each sample was then tested with tenth normal sodium hydroxide using Toepfer's reagent and phenolphthalein as indicators. Free hydrochloric acid and total acid were normal in all of the patients.

V. Lead in Biological Materials (stool, whole blood, and urine)

THE dithizone method was used in the determination of lead in these biological materials. Meticulous care was employed in obtaining the materials and in the estimation of the lead content. The validity of this method was checked by brewing specimens in duplicate and by recovery of known amounts of lead from solutions.

A. Stool Study:

Whenever possible, attempts were made to obtain 24-hour specimens of stool for chemical study. When constipation made it difficult to obtain a spontaneous stool, bowel evacuation was obtained by means of saline cathartics. Specially prepared pyrex containers were employed for the collection of the samples to avoid contamination. The lead content was reported in terms of the number of milligrams of lead estimated in 100 grams of dried stool. Kehoe[28] and his co-workers noted that in the non-industrial population the lead content of 24-hour samples of feces was approximately equivalent to the amounts of lead ingested daily. In a study of the excretion of lead in the stool of normal American adults not exposed to lead hazards, these authors found 0.03 to 0.1 mgm. of lead per 100 gm. of ash feces. Twenty-eight (56%) of this group had normal amounts of lead in the control specimens of stool. In the remaining 22 patients (44%) the amounts ranged from 0.25 mgm. of lead per 100 gm. of dried stool to 4.5 mgm. of lead per 100 gm. of dried stool.

B. Whole Blood:

Wexler and Sobel[29] have established spectographically that the normal values for lead in 100 cc. of serum were below 0.1 mgm. Though the studies were done on a small group of patients with lead poisoning, the validity of the method was checked by recovery of known amounts of lead from solution. Five samples of blood were studied spectographically in our series. The values ranged from 0.1 mgm. of lead to 1.0 mgm. of lead in 100 cc. of serum.

Quantitative lead determinations were made on all of our cases. The blood was drawn in the usual method from the anticubital fossa. Special lead-free needles were used. The syringes and containers were specially prepared to avoid all but the smallest amount of contamination with this heavy metal. Nineteen (38%) of our patients had abnormal amounts of lead in the whole blood. The amounts of lead varied from 0.15 mgm. to 0.75 mgm. of lead in 100 gm. of whole blood. These were chemical estimations by the dithizone method.

We are indebted to Mr. A. E. Sobel and his staff for their cooperation in making these determinations.

C. Urine Studies:

Twenty-four hour specimens (at least one liter) of urine were obtained routinely and studied for lead content. A modified dithizone test was employed by qualified chemists who were thoroughly familiar with the procedure. Special precautions were taken regarding the type of containers which were used for the collection of the specimens. Special care was exercised in the cleansing and preparation of the containers to avoid contamination. The estimation was carried out with great care, to insure accurate results.

Kehoe[30] and his co-workers have established the fact that in the non-industrial American adult, as much as 0.08 mgm. of lead may be excreted in the urine in 24 hours. This group of workers also noted that individuals exposed to the absorption of lead may excrete up to 0.15 mgm. of lead per liter of urine without manifesting any evidence of lead intoxication. Myers[31] and his co-workers, studying the rate of excretion of lead among 205 normal individuals picked at random, noted that an average of 0.033 mgm. of this heavy metal per liter urine, was excreted daily. We have accepted anything in access of 0.10 mgm. of lead per liter of urine as indicating abnormal excretion of lead. Thirty-six (72%) of the patients in our group had abnormal amounts of lead in the urine which was obtained on admission.

Summary

THIS group of 50 patients had a relatively short period of exposure to the absorption of lead. In practically all instances, the inhalation of lead was responsible for the precipitation of acute manifestations of lead poisoning. Seventy-two per cent of the group had evidence of abnormal amounts of lead excreted in the urine, and 38% had an abnormal amount of lead in the whole

blood. There appears to be no parallelism between the lead content of the whole blood and the amount present in the urine.

The presence of stippling of the red blood cells in 40% of the group almost parallels the size of the group in which an abnormal amount of lead was found in the whole blood. Since the excretion of lead is known to occur in tides, it is not unusual to find stippling of the red blood cells in only 40% of the entire group. The presence or absence of stippling varies with the lead excretion tide and is known to vary during the day even among those who are absorbing lead and who do not manifest symptoms of lead poisoning. It is rather difficult from our observations to state that there is any one specific laboratory test which might reasonably be used as an index to indicate or confirm a diagnosis of lead poisoning.

All of our patients were acutely ill and required hospitalization. The laboratory studies were all done during the period of acute lead intoxication. Studies of gastric, hepatic and renal function were done on those patients who presented abnormal amounts of lead in the biological materials. The findings in this group were such as to indicate that despite clinical and laboratory evidences of acute lead poisoning and the presence of an active lead stream, the function of the organ in question was unimpaired.

While one may not be privileged to draw a definite conclusion from this number of studies, it seems evident that, despite the fact that lead bathes all the tissues, there is, even in acute intoxication, no reason to believe that the function of the organ, as stated before, is in any way halted.

Perhaps studies on larger series of cases may show other findings.

However, in these studies we have been impressed with the fact that the liver, which frequently stores lead in its transfer to the skeletal tissue, and the kidney, which excretes lead, are not affected by intoxication with this heavy metal.

Conclusions

WE HAVE recorded the laboratory studies in 50 patients with acute industrial lead poisoning.

2. Blood studies indicated a slight reduction in the hemoglobin paralleled by a reduction in the red blood cell count. The reduction of the platelets, even though slight, was the most constant finding. Basophilic stippling was present in 40% of the group. Polychromasia, lymphocytosis, polynucleosis and eosinophilia were occasionally seen, but with no degree of constancy.

3. There were abnormal amounts of lead in the urine in 72% of the group, in the stool in 44%, and in the whole blood in 38%.

4. Functional tests of the stomach, liver and kidneys were normal.

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Gas Gangrene

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GAS gangrene is a condition which Sacquepee (1920) has defined as follows: "By the name gas gangrene we mean an extremely grave complication of wounds characterized at once by local and general symptoms. The local symptoms are essentially gaseous infiltration, edema, deterioration of the tissues (especially muscular) with gangrenous tendency, and a characteristic smell. The importance of these symptoms varies from case to case. Hence, the manifold types. In addition, various types of erythema (especially bronzed), phytemosis, etc., may be observed. The general symptoms are constant and are marked by extensive poisoning, characterized especially by increased rapidity and extreme weakness of the pulse, dyspnea without thoracic lesion, earthy, sometimes ecteroid color of skin. Unless treated by specific means this very serious affection proves fatal in about 75% of the cases in a few days."

Etiology

GAS infection may complicate any lacerated wound particularly if contaminated by soil, excreta, woolen products, clothes, powder missiles and has been known to follow hypodermic injection, ruptured appendix or criminal abortion. It may be frequently found after compound fracture where injury to muscle and more or less interference with the blood supply favor spread of anerobic infection.

The specific organisms causing gas gangrene are anerobic organisms, alone or in combination, the most important of which are:

1. Bacillus Welchii (B. perfringens, B. aerogenes capsulatus);
2. Vibrion septique of Pasteur (B. of malignant edema);
3. Bacillus oedamatiens (B. novyi, B. bellonensis);
4. Of lesser importance B. fallax, B. sporogenes, and B. histolyticus.

Sacquepee (1920) claims the organisms occur with the following incidence: B. perfringens, 82%; Vib. septique, typical 28%, atypical 11%; B. bellonensis 35%. He concludes that gas gangrene was chiefly due to the above three species of organisms acting alone or in association. Coller and Perham (1937) believe Vib. septique produces more edema but less gas.

The incubation period is from one to four days, even longer, depending upon the virulence of the organism, the amount of devitalized muscle, the number introduced, and the resistance of the host.

Pathology

THE spread of the disease is chiefly in the muscles. It may be confined to a single muscle extending from the wound margin up to its origin, leaving the neighboring muscles free, but usually it spreads to neighboring groups as well, to a varying extent. The muscles become soft, mushy and dark red, and microscopic sections show fragmentation of the muscle fibers and gas bubbles, large numbers of Gram-positive bacilli and a paucity of fixed tissue or wandering cells.

McNee and Dunn (1917) found fascial tissues act as a barrier to the spread of the infection. They feel that the toxins elaborated by the organisms in the wounded muscle caused the death of the adjacent fibers and these were then invaded by the anerobes with the production of gas.

The process of gas production in the tissues is brought about by the saccharolytic ferments elaborated by the bacteria. The gas itself is not toxic, but aids in producing ischemia by pressure upon and occlusion of veins, capillaries and small arteries. Also, gas is not found in detectable amounts in the early periods of an infection. Its presence is a certain proof of a well developed lesion. (Blaustein, 1936.)

Symptoms

EMRYS-Roberts and Cowell (1917) consider three main clinical types in a discussion of the symptomatology of gas gangrene infections:

A. The common type which occurs in neglected wounds 12 to 24 hours after the injury or a few hours after the first operation.

B. The fulminating type, where the patient, if untreated may be dead in a few hours.

C. The delayed type: with a slow onset so that the condition only becomes established several days or weeks after the date of the injury.

The predominating symptoms are those of profound intoxication, high fever, rapid pulse, prostration and apprehension. Locally there is pain in the wound, redness of the skin with swelling, bronzing of the skin and crepitation which is due to the generation of gas by the action of the organisms on muscle tissue. If the wound is open or there are incisions made in the affected tissue, a watery, dirty discharge may be present and a characteristic odor which has been described as "mousy."

Diagnosis

GENTLE pressure on the margins of the wound will produce abnormal tenderness, usually a sanguinopurulent exudate in which gas bubbles may be seen, and crepitation. A smear from the exudate will almost invariably reveal numerous, large Gram-positive bacilli. Gas may also give some aid in diagnosing.

A roentgenogram is of considerable aid in the diagnosis. It is well known that gas in soft tissue may be recognized in a film. It may be due to air introduced by trauma of some sort. If this be the case, the gas shadows will be seen in the immediate vicinity of the wound, and subsequent films will show progressive decrease in the amount, except in a case due to pleural injuries and some sucking wounds near a joint. On the other hand, gas produced by infection tends to increase and

spread away from the wound. The x-ray and clinical picture will vary with the virulence of the organism, a number introduced, length of time, amount of soft tissue injury and contamination, and resistance of the host. If the infection is virulent and involves muscle the muscle fibers may be separated, while on the other hand the collections of gas may be more circumscribed.

X-ray films made a day or two after an operation often reveal an astonishing amount of air trapped in the tissues in the immediate neighborhood of the wound. Care should be taken in diagnosing a postoperative or post-traumatic gas infection from an x-ray examination. Confirmation should be made in a suspected case by examination of the fluid from the wound both by smear and culture. Likewise the appearance of the wound and the characteristic odor are important diagnostic feautres. (Graham, 1936.)

Treatment

THE treatment of gas bacillus infections of the extremities resolves into an early recognition of the symptomatology and the immediate institution of the prophylactic and active therapeutic measures available. The ever mounting number of cases of gas bacillus infections being seen in civil life today may be attributed, to a certain extent, to the hesitancy to utilize proper measures available, procrastinations, or sheer ignorance of the morbid prognosis of crushing injuries of the extremities. The anticipation of complications of any "dirty" wound will serve as a reminder for instituting prophylactic measures." (Davis and Hanelin, 1936.)

The Prophylactic Treatment for gas bacillus infection of the extremities resolves itself into the evaluation as to what wounds are apt to give rise to gas gangrene. After a careful physical examination and history are taken relating to the manner in which the injury was obtained, and eliciting such criteria as whether the patient was injured near soil contaminated by excreta of farm or domestic animals, the examining physician should be on guard for the possibilities of B. Welchii and other anerobic infections. If there is much maceration and crushing of tissue, with or without fracture, an extensive debridement operation is immediately performed under either local or general anesthesia. The wound should be irrigated with oxidizing agents such as hydrogen peroxide, potassium permanganate solution 1:500, Dakin solution, potassium chlorate 2%, and similar solutions.

Intravenous infusion of 2.5% glucose and .45% saline solution will sustain the patient during the immediate shock. External heat is advisable, and morphine, grains one-sixth to one-fourth, should be used as required. The use of commercial prophylactic tetanus and gas gangrene antitoxins is to be highly commended, since the usual dose contains 1500 units tetanus antitoxins, 2000 units perfringens antitoxin and 2000 units Vib. septique antitoxin. This is given in one dose, and usually suffices in the less extensive injuries. However, it may be repeated every other day for three doses, if the judgment of the surgeon and the extent of the injury warrants it. Careful precautions must be used to guard against horse serum reactions, and the usual interdermal tests should be performed before each injection, and any manifestation of anaphylactic reaction should be treated with epinephrine hydrochloride 1-1000 solution.

Hanchett (1934) emphasized the value of effective prophylaxis, debridement and secondary suture in all massive contused and lacerated wounds and compound fracture; polyvalent serum associated with surgery seems to give the best results in his cases.

The Active Treatment of gas gangrene of the extremities must be instituted as soon as the earliest symptoms arise. Glotowa, Ostrawskaja, and Ssilanowa (1935), in an evaluation of gas gangrene sera, state "that in the therapeutic treatment of gas edema disease the perfringens and oedamatiens sera are of especial importance since the B. perfringens as the gangrene stimulus or excitant plays the chief part and the B. oedematiens of more common occurrence accordingly has second place, yet it builds up the most toxic microbic irritation in gangrene and therefore causes the worst course of all the gas edema diseases. But also the Vibrion septique and the B. histolyticus are very significant in gangrene pathogenesis, although much less encountered in the wounds of patients. For these reasons the preparation of excellent curative sera and the possibility of their evaluation are of the greatest importance."

The antitoxin should be given in massive doses, since the toxemia and local tissue reaction is very great. The use of 50,000 to 100,000 units is obligatory. Dickinson and Traver (1937) emphasize the need for polyvalent serum, as many of the cases are of mixed etiology.

The exact Surgical Procedure to follow in any given case is a difficult thing to determine until the presenting symptoms indicate the course of treatment.

Where there is much distortion of anatomical continuity of the structures one is more or less forced into radical procedures to preserve the life of the patient rather than the limb, therefore where there is extensive crushing of the soft tissue parts, accompanied with or without fracture of the bone, a guillotine amputation under scopolomine-morphine and gas anesthesia is indicated. It has been experienced (Davis and Hanelin, 1936) that ether anesthesia has a deleterious effect upon the operative course of these patients. The exact nature of this is hard to determine, but the ether effect appears to be synergistic with the disseminated toxins of the infected organisms. It is advisable to perform a guillotine amputation, high above the level of the spreading infection which can be readily ascertained by the use of the Roentgen ray as pointed out by Davison (1916) and Graham (1936). Secondary prosthesis may be done at a later date when all signs of infection disappear and the patient is entirely recovered from the effects of the toxemia and surgery. Blood

transfusions are indicated at times and should be carried on as frequently as postoperative conditions will permit. The viable stump should be carefully preserved and guarded against secondary trauma, since it has been shown that recurrences of the gas bacillus infections may occur after surgery.

Where there is not much destruction of the tissues due to the injury conservative surgical measures are indicated. Numerous methods of therapy have been advocated, but the essentials—free incision of the wound along the fascial and nerve sheaths, followed by the continuous irrigation of the wound with hydrogen peroxide, potassium permanganate 1:500 or Dakin solution form the basic treatment. Fidean-Green (1934) advocates the use of oxygen gas in the form of continuous irrigation.

The method advocated by Coller and Perham (1937) is a debridement in which all injured and devitalized is removed with scrupulous asepsis. All muscle that has lost the normal color and has lost its contractility, and which does not bleed, must be removed. The wound is then cleansed by mechanical irrigations with bland solutions and if it has undoubtedly been contaminated it is best left wide open to be treated by the Carrell-Dakin method. Appropriate fixation usually of skeletal traction type will give adequate immobilization of bone fragments. Later when the wound is clean and relatively free from infection it can be closed by secondary suture.

When gas bacillus infection is found established in a wound produced by trauma, prompt surgical treatment must be instituted, the character of which will vary with the site and extent of the associated injury. Infections of the superficial parts as the scalp, skin or subcutaneous tissue are not as serious as those involving muscle and are usually free from general systemic reaction. They may be treated by adequate local drainage and dressings utilizing an oxidizing antiseptic. If the circulation of the extremity remains unimpaired, and the infection appears localized to a muscle or muscle group, wide excision of the involved tissue must be carried out. If the circulation of the part is so extensive as to involve all of a part, amputation will be necessary. If such a lesion involves the leg, amputation must be of the guillotine type at the level of the tibial tubercle or better, disarticulation through the knee. In case the thigh is involved in the extensive fashion, a guillotine amputation above the site of the injury must be done with removal of any involved muscle group about this point. The prognosis is poor in all patients with extensive involvement of the thigh. If gas gangrene occur in the stump following a thigh amputation for other cause, reamputation is of no avail and the only operative treatment is wide opening of the wound to receive ample drainage and exposure. All the wounds thus produced should be treated by the Carrell-Dakin method. Unless the infection occur in traumatized muscle, it can be cared for by surgical exposure and drainage.

Eliason, Gilbert, and Erb (1937), after a review of the literature from 1930 to 1936, and in reporting 43 cases of their own, feel that surgery is still the prime indication is most cases of gas gangrene, both as a prophylactic and curative measure. Early debridement of contaminated wounds will protect many patients from the Welch bacillus. They believe the fundamentals in the treatment of solid wounds may be summarized as follows: operate as soon as possible, using nitrous oxide anesthesia, employ longitudinal incisions half again as long as thought necessary, avoid the use of tourniquets and injury to normal muscle, preserving all viable skin, open the wound thoroughly, all torn crushed and discolored muscle to be excised, all loose bone and foreign bodies to be removed, the wound to be left open, filled loosely with moist gauze and the part immobilized. They believe with the aid of serum more conservative surgery can and should be practiced.

Blaustein (1936) believes that in either the prevention or the cure of gas gangrene the treatment is pre-eminently surgical. When a gas bacillus infection has once actively started there is only one acceptable treatment and that is the most radical removal of the infected tissue that is possible to perform. In case of extremities this means an amputation. The stumps are closed loosely; or if there is any doubt whether the operator has gone beyond the involved tissue the stump is left open. It is only in definitely localized infections or in those mild cases with a long period of incubation that radical excision and amputation give place to conservative surgical treatment. He is also of the opinion that the use of a polyvalent serum is an adjunct to both preventive and curative surgical procedures, but is by no means a substitute for them.

Battle Malone (1936) concludes that gas gangrene can be prevented by prompt removal of all contaminated material and tissue from a wound. In this work he has found the gas bacillus antitoxins are of great value, but only as aids to proper surgery. However he also states that the prophylactic serum supplied by manufacturers has not proved its efficacy and that a larger dose of B. Welchii antitoxin is to be preferred.

Newton and Schlappi report the successful treatment of a case of gas gangrene following an attempt to induce abortion, by the use of polyvalent serum and a radical vaginal panhysterectomy.

The use of ether in gas bacillus infection has been a tragedy in the experience of Davis and Hanelin (1936). In two definite cases even before the operation was started the patient died. Since this, 20 years before writing their article, they have not used ether in gas bacillus infections when operating. It is found that the use of scopolamine and morphine with nitrous oxide gas when sawing through the bone has removed this complication of mortality in anesthesia.

Recently the X-Ray Treatment of gas bacillus infections has been reported by Kelly (1933) and Faust (1934 a, 1934 b). Faust states that "there

are several possible theories in the action of the x-rays but as yet no one knows the exact mechanism. But since the results obtained were favorable it is believed the methods of therapy should be given publicity for the good that it may do. One explanation of the action of the x-ray is that when x-rays are played upon nutreint fluids they are deadly to protozoa by producing small quantities of hydrogen peroxide; the presence of hydrogen peroxide would be fatal therefore to the B. Welchii and other anerobic organisms. Due to the absorption of the hydrogen peroxide the x-ray treatments need to be frequent in order to produce more of the gas."

"Another theory of the action of the x-ray is that it causes the tissue to throw out a protein which may be resistant to the toxin of the gas gangrene organisms. The x-ray may act as a catalytic agent by aiding the serum to act on the toxin therapy as given."

The status of the x-ray in gas gangrene is taken up by J. F. Kelly (1936 a). He believes that it is indicated both in limb and trunk, starting as soon as suspected and given throughout the course, twice daily for at least three days. Amputation seems definitely detrimental or at least unnecessary. He concludes that it is safe to rely entirely on gas bacillus serum local antiseptics, local minor surgery and x-ray, since the five deaths in his series occurred in cases with amputation of an extremity, while all those having no amputation survived. Of the 13 patients operated on (amputation), five died and eight recovered, while in the 21 not operated on with extremity involvement, none died. The entire now-amputated group recovered, relying entirely on serum, local antiseptics and small doses of x-ray. Since many patients who had radical surgery still had infected tissue left, it raises the question as to what good surgery does. Then the mortality of those operated on is so high as compared to those not operated on that again the use of amputation seems to be a distinct disadvantage.

The procedure is based entirely on clinical evidence, but Kelly agrees with Faust's (1934) theory of the hydrogen peroxide formation and its action against the anerobes. At any rate, Kelly believes that:

1. X-ray treatments are indicated in all cases of gas gangrene.
2. Treatment should be started regardless of how hopeless the case may seem; and
3. Should be given morning and evening or twice daily and of sufficient voltage to insure penetration of the involved tissue.
4. Finally one should discourage amputation except when tissues are hopelessly damaged by causes other than gas gangrene.

Kelly and Dowell (1936) postulate that the dark appearance shown by the involved tissues does not indicate a true gangrene; it clears up following successful irradiation, and is probably localized cyanosis. The dosage they advocate should be given morning and night for at least three days and of sufficient voltage to insure penetration of involved tissue: 90-100 K. V. on an extremity, 1 mm. of aluminum filter, 130-160 K.V. on trunk with increased filtration; about 100 "r" per treatment over such areas. "Use x-ray in all cases, and serum unless contra-indicated, do not amputate for gas gangrene" says Kelly.

Eyerly (1937) reports a case of gas gangrene following bilateral mid-thigh amputation for Buerger's disease in which considerable improvement was noted following three different exposures to roentgen therapy. The local as well as general condition was markedly improved under this therapy. A definite line of demarcation followed. Temperature dropped to and remained near normal. The pulse rate dropped considerably, from its former high rate. In five days drainage had practically ceased. But due to the considerable area involved, which became necrotic, and sloughed, death ensued.

Eliason, Erb, and Gilbert (1937) received satisfactory results in two cases—the condition disappearing promptly.

Prognosis

THE prognosis is greater with serum as evidenced by a series of Secqueepee (1920) "319 severely wounded morbidity 1%, control 7.2% as a prophylactic agent. As a therapeutic agent Sacqueepee in 191 cases had a mortality of 13%." Eliason, Erb and Gilbert (1937) have collected from the literature (1930-1936) a total of 349 cases, of which 224 were treated with serum, with 56 deaths, a mortality of 25%, and 125 were treated without serum, with 62 deaths, a mortality of 49%.

Conclusion

GAS gangrene is a relatively common complication of contaminated or soiled wounds.

2. It is caused by specific anerobic organisms.
3. Wide destruction of tissue, especially muscular, develops an associated toxemia.
4. Early recognition and prophylactic measures constitute a major part of the treatment.
5. The active treatment resolves into three main types—surgery, x-ray and serum—none of which is specific in itself but best results may be obtained by combinations of the three.

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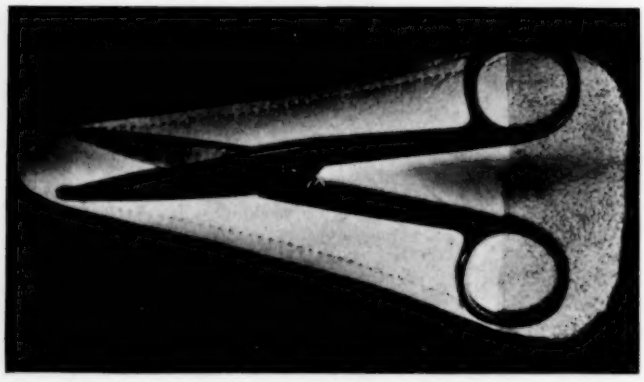
Leather Scissors Carrier

J. ROBERTSON KNOWLES,
Surgeon, Boston & Maine Railroad

I HAVE devised a leather holder for scissors which will be found useful for those physicians who carry scissors in their pocket or in their bag.

It will also be of use to those men who would like to carry a pair of scissors on their person but hesitate in doing so on account of the danger of carrying a sharp-pointed instrument.

The holder is easily made and at very little cost. It consists of two pieces of leather cut out funnel-shape and sewn together. The piece of leather



forming the back wall of the holder should be more or less firm and longer than the one forming the front wall; the piece forming the front wall should be of soft, light, pliable leather.

The accompanying illustration shows the holder with a pair of scissors in place.

Council on Industrial Health

THE second meeting of the Council on Industrial Health was held in the Association headquarters March 26, 1938. Those in attendance were DRS. HARVEY BARTLE, LEROY U. GARDNER, MORTON R. GIBBONS, HENRY H. KESSLER, ALLEN D. LAZENBY, EARL D. OSBORNE, CARL M. PETERSON, CHARLES D. ROBERTS and STANLEY J. SEEGER. DRS. STANLEY J. SEEGER and ALLEN D. LAZENBY were elected chairman and vice chairman respectively.

Other business transacted of interest to physicians and agencies concerned in industrial hygiene was as follows:

1. The Committee on Scope and Early Activities outlined the proposed program of the Council on Industrial Health in these terms:
   - A. Study and invesigation of present activities in the field of industrial health on the part of various organizations and groups.
   - B. Education.
     1. Development of an annual conference to be held in Chicago under the auspices of the American Medical Association.
     2. Contact with state medical societies. The Council feels it desirable to establish contact with the several state medical societies, urging them to create committees on industrial health to assist in the study of problems in this field as they relate to the various communities.
     3. Preparation of special articles. Arrangements have currently been made for papers on syphilis in industry and on silicosis.
     4. Establishment of a clearing house for information in the field of industrial health.
     5. Cooperation with the Council on Medical Education and Hospitals for the fostering of graduate facilities in the field of industrial health.
   - C. Study of medical nomenclature.
   - D. Review of the present compensation laws and the proposals for new laws with special reference to their effect on industrial health.

2. The Committee on Rules submitted regulations regarding policies and procedure, which were adopted.

3. The Committee on Nomenclature reported that it was actively engaged in preliminary work designed to provide the profession with a compendium of terms used in industrial medical practice, integrated as far as possible with the American Medical Association's plans for revision of the Standard Classified Nomenclature of Disease.

4. The Council voted to enlarge its membership and placed in nomination DRS. LEVERETT D. BRISTOL, ANTHONY J. LANZA and CLARENCE D. SELBY. These nominations have been confirmed as regular appointments to the Council on Industrial Health by the Board of Trustees of the American Medical Association.

The survey of present agencies interested in industrial hygiene will include a determination of most suitable means for cooperative effort by the Council on Industrial Health, including the several sections of the Scientific Assembly. It is confidently expected that the influence of the Council on Industrial Health will result in greater emphasis on and interest in occupational health problems, particularly in the sessions of the Section on Preventive and Industrial Medicine and Public Health.

Meeting Calendar

—1938—

JUNE:

American Association of Industrial Physicians and Surgeons, Twenty-Third Annual Meeting, in conjunction with the Midwest Conference on Occupational Diseases, Palmer House, Chicago, Illinois, June 6, 7, 8, and 9, 1938. *Secretary-Treasurer,* Dr. Volney S. Cheney, Armour and Company, Union Stock Yards, Chicago, Illinois.

(The Official Programs for all of the Sessions of both the Annual Meeting of the A. A. I. P. & S. and the Conference appear in full on pages 343 to 347, herein.)

Institute of Traumatic Surgery, June, 1938. *Secretary-Treasurer,* Dr. John D. Ellis, 122 South Michigan Avenue, Chicago.

SEPTEMBER:

American Association of Railway Surgeons, Forty-Ninth Annual Meeting, September 19, 20, 21, Palmer House, Chicago. *Secretary,* Dr. D. B. Moss, 547 West Jackson Boulevard, Chicago.

OCTOBER:

Association of Surgeons of the New York Central System, Fifteenth Annual Meeting, October 24, 25, and 26, 1938, at New York City. *Secretary-Treasurer,* H. A. Fathauer, 413 LaSalle Street Station, Chicago.

NOVEMBER:

Section on Railway Surgery, Southern Medical Association, Oklahoma City, Oklahoma, November 15. 16, 17, and 18, 1938. *Secretary,* Dr. James W. Davis, Davis Hospital, Statesville, North Carolina.

(Program on page 307.)

New York State Society of Industrial Medicine, Eighteenth Annual Meeting, Albany, N. Y., November, 1938. *Executive Secretary,* Frank E. Redmond, 361 Delaware Avenue, Buffalo, N. Y.

DECEMBER:

Association for the Advancement of Industrial Medicine and Surgery, Annual Meeting, New York City, December, 1938. *Executive Secretary,* J. J. Blackford, 370 Lexington Ave., New York.

North Carolina's Mobile X-Ray Laboratory

C. R. Matheson *and* M. F. Trice
Technician and Engineer, respectively,
Division of Industrial Hygiene
of the
North Carolina State Board of Health
and the
North Carolina Industrial Commission

AN indispensable part of the equipment of the Division of Industrial Hygiene is a mobile x-ray laboratory in which pre-employment and routine examinations are made of workers in the dusty trades. A unit of this kind is essential for the satisfactory evaluation of the siliceous dust problem in the state, since the plants involved are widely distributed over a territory that extends 600 miles in an east-west direction and an average of 100 miles from northern to southern boundries.

At many places there are no quarters in which examinations may be made, and at a few locations even electricity is not available, all power being obtained by internal combustion engines. The latter present no insurmountable problems, however, because most operations of this character are situated within a few miles of some town or village where an electrical connection can be obtained. A mobile x-ray laboratory makes possible the examination of employees on the premises of all but a few of the industries involved. This is particularly pleasing to the employers, since the workers lose little more time than is actually required for the examination.

The character and location of the siliceous dust industries provide a conception of the scope of the problem, the study of which is facilitated by the mobile x-ray unit. The foundries, brick and tile plants, granite quarries, and stone-cutting sheds are distributed over the entire state. The mining operations are concentrated largely in the western portion, although several major ones are located in the central area. The mining industries produce feldspar, mica, kaolin, quartz, copper, talc, pyrophyllite, gold, and coal; the several coal mines, however, have been inactive during the past decade. The asbestos textile plants are all concentrated in the central section of the state. In addition to these major categories there is a small group of miscellaneous industries in which the siliceous dust problem is of minor importance.

Prior to the time the mobile laboratory was acquired a portable x-ray unit was transported about over the state on a small trailer drawn by a passenger car. The use of the smaller outfit slowed down the work appreciably, since at each location it was necessary to find suitable quarters and set up the machine. Thus, the time consumed incident to setting up and demounting the equipment was the same at all places regardless of the number to be examined. The

great loss of time occasioned by the almost daily repetition of such work indicated that mobile facilities, while considerably more expensive, would soon justify their cost.

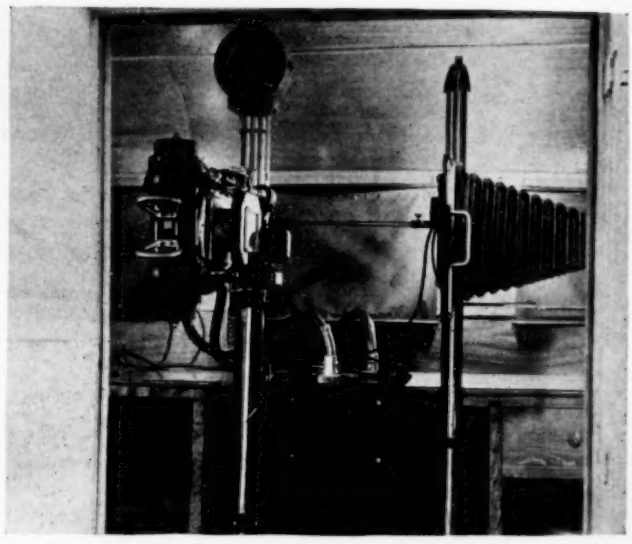
In the two and a half years that the Division has been functioning x-ray photographs of the chest have been made of 6,458 persons, of whom

X-ray trailer and ¾-ton International truck

2,460 were examined in the mibile x-ray laboratory during the past year. These results indicate a lower yearly rate of examination with the new equipment than that established for the original outfit. The mobile unit, however, during the past year has visited small and more remote operations almost exclusively. Much time, therefore, has been consumed in transit between stops. A greater number of the large and more accessible plants were visited during the time that the original equipment was in use, which made possible a greater number of examinations per location.

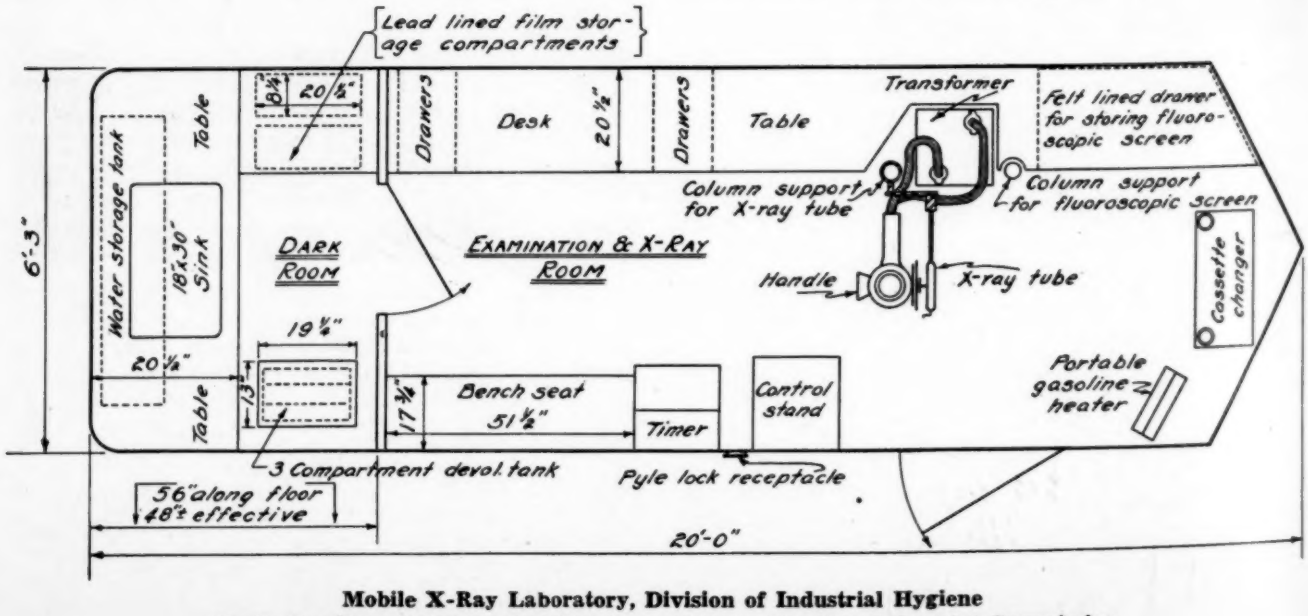
The mobile x-ray laboratory is housed in a 20-foot trailer shell purchased from a manufacturer of a well known line of trailer homes. The trailer is substantially the same as the standard models that are outfitted as living quarters by the builder, differing only in the elimination of a rear door, the construction of a heavier floor, the use of heavier springs, and a rearrangement of windows. The standard inside dimensions—length 20 feet, ceiling height 6 feet 2 inches, width 6 feet 3 inches—remain unchanged.

The interior conforms to plans furnished by the Division and is the work of a local cabinet-maker. A light-proof partition, with a light-proof door, is constructed along a line approximately 4 feet from the rear end to form a small darkroom. A table 20½ inches wide and 36 inches high rests



Scene through doorway showing machine set up for fluoroscopy

against the rear wall of this enclosure. In the center of this table is an 18-inch by 30-inch sink fitted with a plank top, which, in place, provides a table surface that extends the full width of the trailer. Installed on one side of the darkroom is a three-compartment developing tank with inserts that make possible the use of one

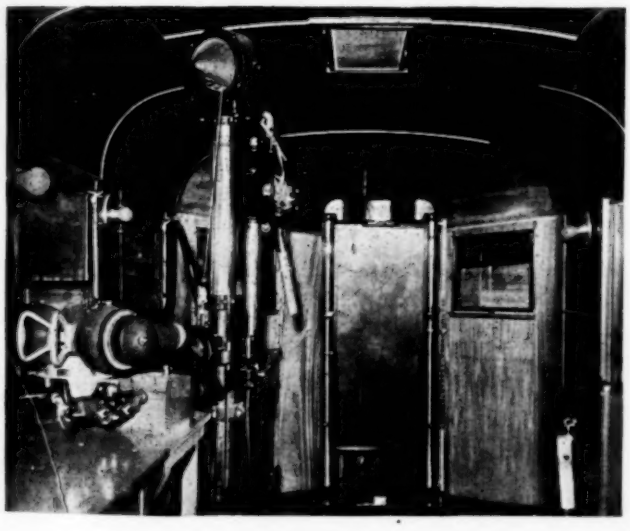


Mobile X-Ray Laboratory, Division of Industrial Hygiene
North Carolina State Board of Health and North Carolina Industrial Commission

to three gallons of solution. On the other side, lead-lined film storage compartments are built into a cabinet which is provided with a plank top to extend the work table area to two sides of the room.

Along one wall of the examination and x-ray room a table 20½ inches wide and 30 inches high extends from the partition to the front end of the trailer. Under this table, near the partition, and over the wheel housing are two tiers of drawers, built in to form a desk. Near the front end of the trailer this table contains a large felt-lined drawer that accommodates the fluoroscopic screen. The remaining space under the table is occupied by a transformer, columns that support the x-ray tube and fluoroscopic screen, and collapsible chairs and other miscellaneous equipment. Running along the opposite wall, over the wheel housing, is a small bench 15 inches wide, 51½ inches long, and 16 inches high which will accommodate several workers awaiting examination.

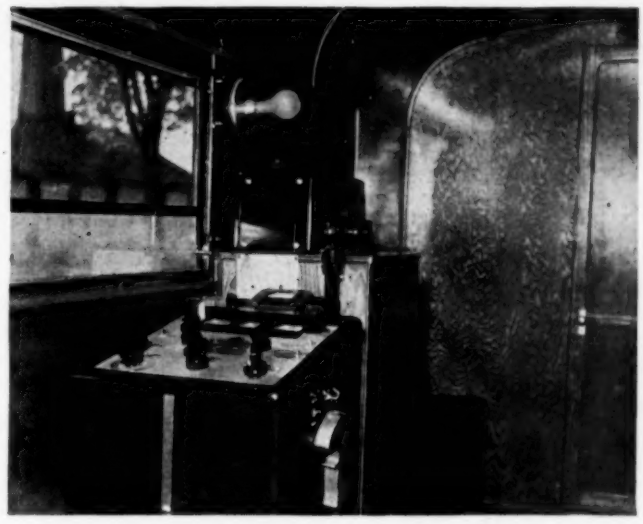
The laboratory contains only a small amount of plumbing. Strapped to the floor under the sink in the darkroom is a 30-gallon galvanealed metal water-storage tank which serves the developing tank and one spigot, mounted over the sink. Two lengths of garden hose and suitable



Interior view of trailer showing x-ray tube bolted down to table for traveling. Plate changer appears in the front end

tank fittings permit connection to a local water system. The sink and developing tank drain pipes terminate a few inches below the trailer floor.

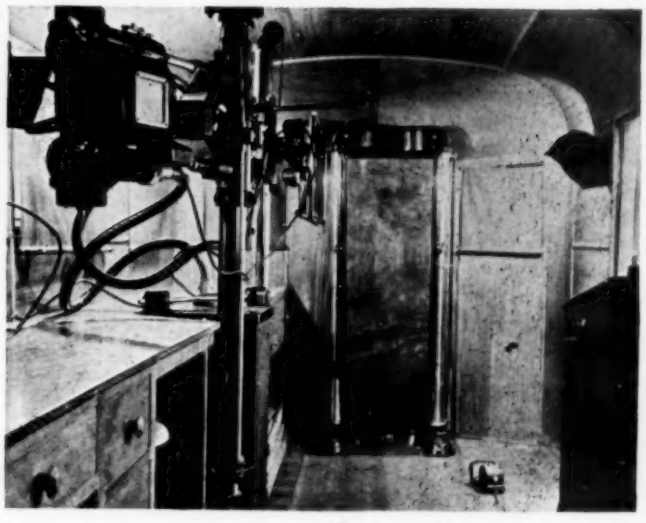
Prior to the installation of the present x-ray equipment the original portable x-ray outfit was used in the house trailer for a period of approximately six months, during which time it became evident that a larger machine was desirable. The trailer was purchased primarily to provide examination quarters. Had it been possible to foresee the acquirement of a larger x-ray unit, a trailer specially designed to accommodate such equipment would have been procured. Under the circumstances it became necessary to utilize existing facilities to the best advantage.



Interior view of trailer showing control panel and timer. In the rear along the wall is a seat for persons awaiting examination

The x-ray and fluoroscopic equipment is mounted in the trailer in a manner to provide the maximum space for physical examinations, due consideration being given to an advantageous distribution of weight.

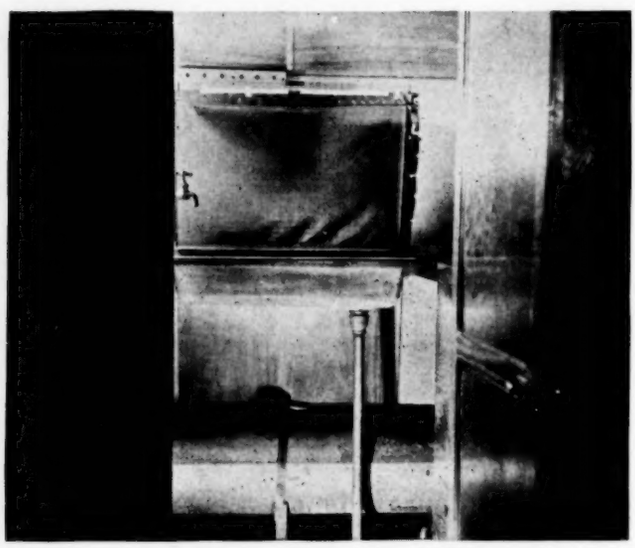
The control stand, the timer, and a bench are located on one side of the front compartment. The control stand is near the entrance and is mounted on shock-absorbing rubber cushions that are anchored firmly to the floor. This control panel differs somewhat from the conventional type in several respects. It contains a line voltage regulator that will compensate for a



Interior view of trailer showing x-ray tube and fluoroscopic screen swung back over tables to allow free passage when using the trailer as office space

170- to 250-volt variation. A volt meter is available to use with the line voltage regulator. A lock plug connection at the side makes possible a 110-volt current when it is impossible to obtain it by grounding and fusing one side of the 220-volt line. There is a toggle switch for cutting out all filament circuits when the 110-volt trailer

illumination current is taken from the auto-transformer of the machine. Another toggle switch permits changing from large to small x-ray tube focal spot and vice versa. Finally, there is a more sensitive x-ray filament regulator for pre-setting the milliamperage. Just back of the control stand is the impulse timer, which is placed upon the top of the cabinet housing the main relays and contactors. The timer has a range of 1/120- to 1/5-second exposure and incorporates a device for making a visual check of each exposure. Extending from the timer to the darkroom partition is the previously mentioned bench.



Interior scene through door to darkroom showing sink and pressure tank. The sink is built into a table which extends the width of the trailer and is provided with a demountable plank top which, when in place, permits the sink to be used as a work table. On the left behind the partition is a three-gallon capacity developing tank, and behind the partition on the right is a lead-lined film storage chest

On the other side of the trailer and almost directly in front of the entrance are the x-ray tube and its supporting column, the transformer, and a supporting column for the detachable fluoroscopic screen, which, incidentally, is of a type that permits daylight fluoroscopy. The two columns have metal bases which are held in position by metal collars that are welded to a steel floor plate which, in turn, is bolted to the steel frames of the trailer. The x-ray transformer is located between the upright columns. It is mounted on shock absorbing rubber cushions bolted to the same metal floor plate. The transformer utilizes space between the columns that otherwise would be wasted and permits the use of short flexible cables to the x-ray tube. It has a capacity of 200 milliamperes at 89 Kv.P with full wave valve tube rectification. These tubes are oil immersed and are contained in the transformer case.

A vertical cassette changer is located in the extreme front end of the trailer. It is placed slightly off-center to give access to the long table drawer that carries the fluoroscopic screen. In addition, its position results in a more balanced distribution of weight. The cassette changer is bolted to the floor, which is reinforced on the under side by a metal plate.

Electrical connection to a local source of power is made through a waterproof Pyle lock receptacle which is installed in the side of the trailer near the floor level at a point just back of the control stand. A 220-volt, 60-cycle, alternating current is required, which is obtained by direct connection to the switch box of the plant under study by means of a 125-foot weatherproof rubber insulated, 2-strand, No. 0 cable. By grounding and fusing one side of the 220-volt line a 110-volt current for lighting the trailer may be obtained. In view of the limited space in the trailer and the fact that an overload safety release is part of the equipment, a cutout switch for the main line has been temporarily omitted. The cables from the control stand to the transformer are inclosed in a conduit pipe underneath the trailer. The x-ray and fluoroscopic equipment is shockproof throughout. In transit the x-ray tube arm is bolted to the table top between two blocks of wood that are cushioned with rubber matting. The fluoroscopic screen is carried in the felted drawer already mentioned.

The darkroom is used primarily for loading cassettes; only trial films and those needed for immediate diagnosis are developed in the field. The bulk of such work is done in the Division offices in the State Board of Health Building.

The trailer is drawn by a ¾-ton truck on which a special body has been built to accommodate accessory equipment. In a front compartment are two reels that carry the electric cable and the garden hose, respectively. A middle compartment is reserved for a card index file which provides readily available information on previous examinations and prevents duplication of effort. A rear compartment accommodates a spare tire.

From the Archives

SICKNESS is a far greater cause of industrial inefficiency, spoilage, absenteeism, labor turnover, and financial loss in general than injuries.* It is because of this economic factor that industry will more and more assume the financial responsibility for the health of its employees.

Industrial medicine is in reality a phase of public health. As a leading authority has recently stated (Winslow), "Industrial medicine has vast possibilities of usefulness, and in this country has already accomplished more than any of the other tendencies which have been noted in bringing medical service to the individual at a stage when the most effective measure of prevention can be assured."

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* From a paper by C. O. SAPPINGTON, M.D., DR. P.H.; "Sickness Versus Accidents in Industry." *California & Western Medicine*, April, 1928, Vol. XXVIII, No. 4; Read before the California Medical Assn., Industrial Medicine and Surgery, Section, at the Fifty-Sixth Annual Meeting, April 25-28, 1927.

Medicolegal Trends: Occupational Diseases

— A Statistical and Legal Analysis of Medicolegal Experience —
— (1938 Report) —

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Consulting Industrial Hygienist,
Chicago, Illinois

THE medicolegal problems of occupational diseases have come to be of paramount importance to a great many different groups of people, including employees, employers, legislatures, industrial commissions, courts, as well as the professional groups of physicians, attorneys, and engineers.

Because of rapid changes in legislation and wide differences in compensation experience in various states, it is essential to make periodic statistical reviews, in the endeavor to estimate the trends in this ever changing field.

It is well to repeat that any analysis of the medicolegal trends of occupational diseases involves a consideration of all of the aspects of occupational disease problems, including the measurement and evaluation of industrial exposures; the interpretation and application of information relating to physical examinations, diagnoses, clinical laboratory work, and x-ray findings; the correlation of the industrial and medical information in terms of cause-and-effect relationships; occupational disease legislation; case decisions of damage suits, commission hearings, and review decisions; and insurance coverage.

Using recent material available, it is desirable in this discussion to consider experiences with occupational disease claims and reporting; the cost of compensation for occupational diseases; comparison of older experiences with more recent ones; a brief analysis of higher court decisions during 1936 and 1937; and finally, brief reference to present types of statutory coverage.

Disease Claims and Reporting Experiences

RATHER extensive data have been compiled by the State Department of Labor of New York, especially with reference to the incidence of disease claims and the types of claims closed during the last two years. The original data for 1937 although unpublished at this writing, have been supplied through the courtesy of the Executive Director of the Industrial Hygiene Division, Dr. Leonard Greenburg, and these figures have been rearranged in tables especially for this paper, on the basis of frequency, and in certain instances where expedient, percentage figures have been added.

Table I shows the disease claims closed in the State of New York during the years 1936 and 1937. Note that while in 1936 there was a total number of 1743 claims closed, this increased in 1937 to 2675, or an increase of 932 cases, equivalent to an increase of 53.5% in the total number of cases closed. This table also shows that there were 684 awards in 1936 as compared with 1125 in 1937, an increase of 441 or 64.4%; with reference to the claims which were denied, there were 1059 in 1936, as compared with 1550 in 1937, an increase of 491 cases, or 46.3%. The figures show further that there was a slight increase in the number of cases awarded in 1937 as compared with 1936, 42% and 38% respectively. There was of course a corresponding decrease in the number of cases denied.

Table I.
DISEASE CLAIMS CLOSED
STATE OF NEW YORK — 1936 AND 1937

	All Cases	%	Awards	%	Denied	%
1936	1743	100.0	684	38.0	1059	62.0
1937	2675	100.0	1125	42.0	1550	58.0
Increase in number of cases	932	53.5	441	64.4	491	46.3

Because reasons for the denial of claims are of importance in medicolegal relationships, a tabulation has been made of these in Table II. It will be noted in this experience for 1937 that a separation has been made of dermatitis claims and other claims; that there were 1550 cases in all denied, of which 883 or 56.8% were dermatitis claims and 667 or 43.2% due to other causes. There were four chief causes for denial of claims: "No lost time (30.9%)—"Not over 7 days disability" (19.0%)—"Not covered by law" (15.8%)—and "Failure to prosecute" (10.2%). The other reasons for denial

Table II.
DISEASE CLAIMS CLOSED
STATE OF NEW YORK — 1937.
REASONS FOR DENIAL

Reason	Total No. Cases	Total %	Dermatitis No. Cases	Dermatitis %	Other Claims No. Cases	Other Claims %
ALL CASES	1550	100.0	883	56.8	667	43.2
No lost time	480	30.9	368	76.6	112	23.4
Not over 7 days disability	294	19.0	176	59.8	118	40.2
Not covered by law	245	15.8	57	23.2	188	76.8
Failure to prosecute	159	10.2	105	66.0	54	34.0
No medical evidence	128	8.3	66	51.6	62	48.4
Non-appearance of claimant	103	6.1	60	58.3	43	41.7
Not proved	67	5.2	27	40.3	40	59.7
No causal relation	43	2.8	13	30.2	30	69.8
Unspecified	5	0.3	0	0.0	5	100.0
Withdrawn	4	0.2	1	25.0	3	75.0
Non-cooperative	4	0.2	2	50.0	2	50.0
No lost earnings	3	0.2	0	0.0	3	100.0
Overtime limit	3	0.2	0	0.0	3	100.0
No jurisdiction	3	0.2	2	66.7	1	33.3
No case established	2	0.1	0	0.0	2	100.0
Occupation not covered	1	....	0	0.0	1	100.0
Triparty action	1	....	1	100.0	0	0.0

* Paper presented before the Annual Meeting of the Central States Society of Industrial Medicine and Surgery, at Springfield, Illinois, May 17, 1938. (NOTE: Presented with lantern slides.)

occurred in smaller percentages, but are set forth in detail so that the entire group of causes for denial of claims may be available for reference purposes.

Table III.
DISEASE CLAIMS CLOSED
STATE OF NEW YORK — 1937

Type	Cases	%	Awards	%	Denied	%
ALL CASES	2675	100.0	1125	42.0	1550	58.0
Dermatitis	1564	58.6	681	43.6	883	56.4
Poisons	176	6.6	82	47.6	94	52.4
Lead	82	3.1	42	52.5	40	47.5
Carbon monoxide	40	1.4	15	38.6	25	61.4
All lung conditions	156	5.8	30	19.3	126	80.7
Tuberculosis	56	2.1	3	5.3	53	94.7
Silicosis	21	0.8	9	42.7	12	57.3
Bronchitis	19	0.7	6	31.6	13	68.4
Pulmonary lesion	16	0.6	1	6.2	15	93.8
Pneumonia	14	0.5	1	7.1	13	92.9
Asthma	9	0.3	1	11.1	8	88.9
Pneumoconiosis	5	0.2	3	60.0	2	40.0
Silicosis and Tuberculosis	4	0.1	2	50.0	2	50.0
Pleurisy	4	0.1	0	0.0	4	100.0
Synovitis	139	5.2	90	64.8	49	35.2
Infection	86	3.2	54	62.8	32	37.2
Bursitis	72	2.7	35	48.6	37	51.4

Table III shows the disease claim experience in the State of New York for 1937, giving the principal reasons for the filing of claims; this table is not complete, but merely shows the principal causes of claims (except for the rather detailed analysis under "All Lung Conditions"). Dermatitis again heads the list for 58.6% of all causes. Poisons which include principally lead and carbon monoxide accounted for 6.6%. All lung conditions came third with 5.8%, with the detailed analysis as shown. Synovitis was responsible for 5.2% of the claims, infections for 3.2% and bursitis for 2.7%.

Table IV.
DERMATITIS CLAIMS CLOSED
STATE OF NEW YORK — 1937
(By Industry or Occupation)

Industry or Occupation	Cases	%	Awards	%	Denied	%
ALL CASES	1564	100.0	1125	42.0	1550	58.0
Restaurant	183	11.7	97	53.0	86	47.0
Machinery	78	5.0	30	38.4	48	61.6
Realty	72	4.6	33	45.8	39	54.2
Garment	65	4.1	21	32.3	44	67.7
Printing	63	4.0	20	31.8	43	68.2
Cosmetics	61	3.9	31	50.8	30	49.2
Fur	60	3.8	28	46.7	32	53.3
Construction	58	3.7	30	51.7	28	48.3
Mercantile	44	2.8	14	31.8	30	68.2
Hotel	39	2.5	20	51.3	19	48.7
Paint	35	2.2	15	42.8	20	57.2
Soda fountain	33	2.1	18	54.5	15	45.5
Shoe	33	2.1	22	66.7	11	33.3
Automobile	30	1.9	11	36.7	19	63.3
Hospital	24	1.5	15	62.6	9	37.4

Because dermatitis greatly outnumbered all causes for claims, Table IV was devised to show the industries or occupations which contributed materially to this type of claim. Except for the restaurant group, showing 11.7% of dermatitis claims closed during 1937 in the State of New York, there is a fairly even spread over the different industries and occupations as noted. The original data from which this table was made disclosed the fact that the chief causes of dermatitis claims were soap and water and that dyes of various kinds occupied the next place in point of frequency. The percentage distribution of the claims, therefore, by industry or trade as noted in Table IV results on the whole from exposure to these substances.

A classification of disease awards according to the state compensation law schedule is shown in Table V. Again, dermatitis leads (although somewhat masked by inclusion under other headings) while "Any and all occupational diseases," "Bursitis and synovitis," "Blisters and abrasions," and "Benzol poisoning and sequelae" follow in order. Attention is particularly called to the fact that there were no awards for the various diseases or substances as shown in the note at the bottom of the table.

Table V.
DISEASE CLAIMS CLOSED
STATE OF NEW YORK — 1937
AWARDS BY PARAGRAPH OF SCHEDULE LAW

Paragraph	Disease or Condition	Number of Cases: Awards	Percent
	ALL CASES	1125	100.0
1	Anthrax	6	0.5
2	Lead poisoning and sequelae	43	3.8
4	Mercury poisoning and sequelae	6	0.5
7	Wood alcohol poisoning	3	0.3
8	Benzol poisoning and sequelae	61	5.4
	Aniline and others, and sequelae	20	1.8
9	Carbon bisulphide, sequelae	1	.....
	Any sulphide	4	0.4
12	Dope poisoning, sequelae	1	.....
	Carbon tetrachloride, sequelae	2	0.2
13	Formaldehyde poisoning	8	0.7
14	Chrome ulceration, sequelae	.....	.....
	Chromic acid	14	1.2
	Bichromates	6	0.5
22	Carbon monoxide poisoning	15	1.3
23	Acids, sulphuric	2	0.2
	hydrochloric	4	0.4
25	Blisters, sequelae	70	6.2
	Abrasions, sequelae	13	1.1
26	Bursitis	35	3.1
	Synovitis	90	8.0
27	Dermatitis, acids	36	3.2
	alkalis	256	22.8
	oils	134	11.9
28	Any and all occupational diseases (Sec. 3)	279	24.9
4-A	Silicosis (Sec. 66)	15	1.3
	Unspecified disease	1	.....

(No awards for (3) Zinc; (5) Phosphorus; (6) Arsenic; (10) Nitrous fumes; (15) Skin cancer; (16) Glanders; (17) Compressed air; (18) Miners' diseases; (9) Cataract in glass workers; (20) Radium; (21) Methyl chloride; (24) Petroleum products; during 1937.)

Referring now to the experience in the State of Ohio, there are available data for the compensable occupational diseases reported during the 10-year

period, 1928-1937 inclusive as shown in Table VI. Of a total of 12,931 cases of compensable occupational diseases, dermatitis again appears in first place with 8,730 cases or a percentage of 67.5; lead poisoning with 1,464 cases or 11.3% was second; tenosynovitis was third with 1,452 cases or 11.2%.

Table VI.

Summary of Compensable Occupational Diseases Reported during 1928-1937, Inclusive
State of Ohio

	Number	Percent
All Cases	12,931	100.0
Dermatitis	8,730	67.5
Lead poisoning	1,464	11.3
Tenosynovitis	1,452	11.2
Chrome ulceration	390	3.0
Silicosis	220	1.7
Prepatellar bursitis	212	1.6
Compressed air illness	170	1.3
Benzol poisoning	68	0.5
Brass or zinc poisoning	56	0.5
Volatile petroleum products poisoning	52	0.4
Carbon bisulphide poisoning	34	0.3
Sulphur dioxide poisoning	25	0.2
Potassium cyanide poisoning	18	0.1
Arsenic poisoning	15	0.1
Epithelioma (skin or eyes)	14	0.1
Wood alcohol poisoning	5	.....
Carbon dioxide poisoning	3	.....
Phosphorus poisoning	2	.....
Anthrax	1	.....

(No cases of Glanders, Mercury poisoning, Manganese dioxide poisoning, or Radium poisoning reported 1928-1937, inclusive.)

It seems significant that no cases were reported during this 10-year period relative to glanders, mercury poisoning, manganese dioxide poisoning, or radium poisoning. It should be pointed out, however, that this is mentioned only for the State of Ohio, and one may not conclude that it is of real significance for other states.

In the State of Illinois, the new occupational disease disability compensation law went into effect on October 1, 1936. During the first year of the operation of this law, there were 220 claims filed, as detailed in Table VII. Lead poisoning leads with 47 cases (21.7%), while silicosis with 15% and pulmonary tuberculosis with 10.9% were among the leading causes given for filing of claims. Noting all the pulmonary conditions alleged in these claims filed, one finds a total of 84 cases, or a percentage of 26.2 of the total number filed.

Table VII.

Occupational Disease Claims Filed
(First Year of O.D. Disability Compensation Law, 1936-37)
State of Illinois

Complaint	No. Claims	Percent
All Cases	220	100.0
Lead poisoning	47	21.4
Indefinite complaints	41	18.6
Silicosis	33	15.1
Miscellaneous	28	14.1
Tuberculosis, pulmonary	23	10.9
Dermatitis	20	9.1
Pulmonary diseases	14	6.4
Silicosis and tuberculosis	11	5.0
Asbestosis	3	1.4

In Connecticut, it has been possible during recent years to compare the relationship between the cases of occupational diseases reported by physicians and those cases which have been compensated but not reported by physicians. The significant differences occurring under these two types of classification are shown in Table VIII. It is noted for instance that all cases reported by physicians amounted to 127, whereas all cases compensated but not reported by physicians numbered 286—during the fiscal years July, 1936, to June, 1937. These figures clearly show no correspondence whatever between the cases reported by physicians and those compensated but not reported. The State of Connecticut is now attempting to correct by appropriate legislation and administration this apparent incoordination of the reporting system with compensation activities.

Table VIII.

Relation of Cases Reported to Cases Compensated Occupational Diseases
State of Connecticut — July, 1936 — June, 1937.

	Reported by Physicians	Compensated–Not Reported
All Cases	127	286
Dermatitis	68	154
Friction and tension diseases	0	50
Silicosis and pneumoconiosis	35	4
Lead and lead compounds	9	4
Carbon monoxide poisoning	1	11

Cost of Compensation

THE cost of compensation for occupational disease disability has always been an interesting subject to industrial executives and insurance companies. While statistical material on this subject is somewhat meager and not by any means standardized as yet, the cost figures which have been published by the State of Wisconsin are probably the best that can be secured at the present time. Wisconsin has had an occupational disease disability compensation law longer than any other state and has, therefore, had an opportunity to have more experience with the administration of the law and the payment of compensation than

Table IX.

Compensation Paid for Occupational Disease Disability
Wisconsin — 1936

Classification	No. Cases	Total Indemnity Paid	Average Indemnity Paid Per case
All Cases (this group)	1069	$215,990	$ 202
Noxious dusts	71	119,737	1,686
Vapors, gases, fumes	90	32,709	363
"Germs"	31	2,858	92
Toxic fluids	824	57,580	70
Miscellaneous irritants	153	3,106	20

Indemnity paid includes only compensation paid—does not include other expenses.

any state in the country. This long experience makes comparisons of previous figures and more recent ones in this state, more significant.

The most recent figures available from Wisconsin are for the year 1936 and the report on these data was released in November, 1937. Observe in Table IX that in the classification of the principal types of occupational disease, the total indemnity paid for all cases of this group numbering 1069 was $215,990, or an average compensation per case of $202. Of particular significance is the fact that noxious dusts with 71 cases, or 6.6% of the total number, caused payment of compensation of $119,737 or 55.4% of the total indemnity paid for the group of diseases as noted. The average compensation cost for noxious dust diseases was, therefore, high, being $1,686 as compared with the next highest of $363 for vapors, gases and fumes.

Table X.
COMPENSATION PAID FOR O. D. DISABILITY
COMPARISON OF 1936 WITH 1920-1929, INCLUSIVE
STATE OF WISCONSIN

CLASSIFICATION	No. Cases 1936	Avg. No. Cases Per Year 1920-29	Average Indemnity Per Case 1936	Average Indemnity Per Case 1920-29
ALL CASES (this group)	1069	302	$ 202	$191
Noxious dusts	71	40	1686	298
Vapors gases, fumes	90	35	363	414
"Germs"	31	20	92	322
Toxic fluids	824	84	70	52
Miscellaneous irritants	153	32	20	47

Total amount of indemnity paid 1920-1929 inclusive—$575,052

Total amount of indemnity paid in 1936—$215,990.

Table XI.
SILICOSIS CLAIMS CLOSED DURING 1936
WISCONSIN

INDUSTRY	No. Cases	Working Days Lost: Total	Working Days Lost: Average Per Case	Indemnity: Total Paid	Indemnity: Average Paid Per Case
Machinery mfg.	6	34,800	5,800	$41,992	$6,998
Iron mining	4	24,000	6,000	21,192	5,298
Quarrying	3	13,800	4,600	15,484	5,161
Stone cutting and polishing	2	12,000	6,000	12,859	6,429
Gray iron foundry	2	3,800	1,900	6,609	3,304
Enameled iron ware	2	12,000	6,000	4,758	2,379
Motorcycle mfg.	1	6,000	6,000	7,378	7,378
Furnace	1	1,500	1,500	1,585	1,585
Malleable iron foundry	1	885	885	3,093	3,093
ALL CASES	22	108,785	4,944	$114,950	$5,225

Indemnity does not include medical, cost of hearing, or other expenses.

Average lost time per case represents the loss of 16.4 working years per case or an aggregate of 360.8 working years for the 22 cases.

It is of interest to make a comparison of available figures relative to the cost of compensation in the State of Wisconsin during the decade of 1920-1929 inclusive (in the earlier years of the operation of the law) with the figures available for the year 1936. Table X shows that the average number of cases per year during the above mentioned decade was 302, while the number of cases for 1936 was 1069. The current figures show that whereas the total amount of indemnity paid for the diseases as noted in this group was $215,990 in 1936, the average amount paid annually for the same group of diseases during the decade 1920-1929 inclusive was $57,505. The average compensation costs for dust cases therefore increased more than five-fold, while the total number of cases has increased over three-fold.

Table XI shows the detail for silicosis claims closed in Wisconsin during 1936. Note that there were 22 cases in all, causing an average loss of 4,944 working days per case and an average compensation cost of $5,225 per case.

Table XII.
COMPARISON OF SPECIFIC OCCUPATIONAL DISEASE EXPERIENCE
WISCONSIN — 1936 (CLAIMS CLOSED)

DISEASE	No. Claims	Working Days Lost: Total	Working Days Lost: Average Per Case	Indemnity: Total Paid	Indemnity: Average Paid Per Case
Lead poisoning	13	561	43	$ 1,373	$ 105
Dermatitis	150	3,221	21	7,780	51
Silicosis	22	108,785	4944	114,950	5,225

It was thought advisable in working with the Wisconsin statistics to compare the working time lost, and the cost of compensation of the silicosis cases, with other principal types of occupational diseases. Accordingly in Table XII these differences are shown with regard to lead poisoning, dermatitis and silicosis. The figures clearly show that there is a disproportionate time loss and compensation cost for silicosis cases, as compared with cases of lead poisoning and dermatitis.

On further examination of the cost figures relative to the 1936 figures in Wisconsin, it was discovered that of the 22 cases of silicosis closed, 11 were fatal cases. It was, therefore, thought desirable to compare the fatal cases of silicosis with all types of fatal cases caused by serious injuries, as shown in Table XIII. It will be observed that the average compensation cost of the fatal cases of silicosis stood second to the average compensation cost of the cases of electrocution, $4,753 and $5,243 respectively.

Table XIII.
FATAL CASES CLOSED IN 1936
WISCONSIN

Injury	No. Cases	Total Indemnity Paid	Average Indemnity Per Case
ALL CASES	169	$696,946	$4,123
Fractures	69	272,741	3,953
Crushing injuries	27	107,098	3,966
Electrocution	11	57,675	5,243
Silicosis	11	52,283	4,753
Burns or scalds	13	49,655	3,819
Cuts or punctures	6	27,573	4,595
Asphyxiation	5	14,841	2,968
Septicemia	3	13,717	4,572

Finding such severity in the silicosis cases in Wisconsin during 1936, it was considered advisable further to make comparisons of the permanent

total disability cases and also of the so-called relative permanent partial disability cases during the same period. These comparisons are set forth in Tables XIV and XV. Table XIV shows that of the seven permanent total disability cases closed in Wisconsin during 1936, silicosis accounted for five and averaged the highest compensation cost of $7,815.

Table XIV.
PERMANENT TOTAL DISABILITY CASES CLOSED IN WISCONSIN — 1936

Injury	No. Cases	Total Indemnity Paid	Average Indemnity Per Case
ALL CASES	7	$49,142	$7,020
Silicosis	5	39,076	7,815
Concussion of brain	1	6,801	6,801
Fractured femur	1	3,265	3,265

In Table XV the data are not complete, because merely the principal causes of the relative permanent partial injury cases have been included. Observe, however, that silicosis was again second in the average compensation paid per case ($3,932) while burns and scalds accounted for the exceedingly high average compensation cost of $8,705 per case.

Table XV.
RELATIVE PERMANENT PARTIAL INJURY CASES CLOSED IN WISCONSIN IN 1936

INJURY	No. Cases	Working Days Lost: Total	Working Days Lost: Average Per Case	Indemnity: Total Paid	Indemnity: Average Paid Per Case
ALL CASES (in this special grouping)	67	85,518	1,276	$227,887	$3.401
Fractures	29	35,023	1,208	94,053	3,243
Bruises and abrasions	15	17,400	1,160	53,111	3.541
Sprained or strained back	6	3,750	625	7,980	1,330
Silicosis	6	12,785	2,131	23,591	3,932
Cuts, punctures, lacerations	5	5,520	1,104	18,663	3,733
Concussion, shock	4	5,190	1,298	12,080	3,020
Burns, scalds	2	5,850	2,925	17,409	8,705

Table XVI.
HIGHER COURT DECISIONS — 1936 (42)

SILICOSIS	18
(Wis. 8 — Mo. 2 — N. Y. 2 — N. C., Ill., W. Va., Penn., Ga., U. S. — one each.)	
PNEUMOCONIOSIS	5
(N. Y. 2 — Ill. 2 — Mo. 1)	
TUBERCULOSIS	5
(Wis. 2 — Minn., Conn., Mo. — one each)	
LEAD POISONING	2
(Tenn. — Mo.)	
DERMATITIS	2
(Mass. — Ore.)	
MISCELLANEOUS	10
(Conjunctivitis, Mo. — Manganese poisoning, N. J. — Pneumonia, Ill. — Radium, N. J. — Chlorine, Mich. — Skin Cancer, U. S. — Benzol, N. Y. — Asbestosis, N. C. — Bronchitis, Miss. — Emphysema, Mass.)	
(All lung conditions	33)

Higher Court Decisions

HIGHER court decisions reflect the incidence of appealed cases either from commission rulings or from lower court rulings. The details of this experience are shown in Table XVI for 1936 and in Table XVII for 1937. Note that in 1936 there were a total of 42 published decisions rendered by the higher courts, of which 18 were assignable to silicosis and other chief causes as shown. If we combine all lung conditions, we find that there were a total of 33 decisions involved.

Table XVII.
HIGHER COURT DECISIONS — 1937 (44)

SILICOSIS	15
(Wis. 5 — N. J. 2 — Calif. 2 — Minn., Ga., N. Y., Penna., Nebr., Ill. — one each)	
TUBERCULOSIS	7
(Wis. 2 — N. Y. 2 — Mo., Ill., N . C. — one each)	
LEAD POISONING	6
(Mo. 2 — Nebr. 2 — Idaho, N. Y. — one each)	
PNEUMOCONIOSIS	4
(Kas., Ark., Ill., Mo. — one each)	
DERMATITIS	2
(Illinois and Maine — one each)	
MISCELLANEOUS	10
(Tenosynovitis, Ariz. — Phosphate poisoning, Ga. — Foot ulcers, Wis. — Pneumonia, Texas — Benzol poisoning, N. Y. — Chlorine, Wash. — Asbestosis, N. C. — Poliomyelitis, Calif. — Chrome ulceration, Mich. — Carbon monoxide, Okla.)	
(All lung conditions	29)

The higher court decisions in 1937 as shown in Table XVII show that there were 44 decisions rendered, of which silicosis was responsible for 15. "All lung conditions" totaled 29 decisions.

Table XVIII.
TYPE OF STATUTORY COVERAGE FOR OCCUPATIONAL DISEASES (By States) — 1938

I. "BLANKET" COVERAGE	8
Mo., N. D., Calif., Conn., Ill., Ind., Mass., Wis.	
II. SCHEDULE COVERAGE	13
Del., Ky., Mich., Minn., Nebr., N. J., N. Y., N. C., Ohio, Penna., R. I., Wash., W. Va.	
III. No COVERAGE	27
Ala., Ariz., Ark., Colo., Fla., Ga., Idaho, Iowa, Kas., La., Me., Md., Miss., Mont., Nev., N. H., N. M., Okla., Ore., S. C., S. D., Tenn., Texas, Utah, Vt., Va., Wyo.	
(These states, with Del., Minn., N. J. and R. I., make no provision for coverage of dust diseases.)	

Existing Compensation Laws

THE kinds of statutory coverage for occupational diseases (by states) existing in 1938 are set forth in Table XVIII. There are two types of coverage as shown by this table — so-called "blanket" coverage which includes states which have a law providing that there is general coverage for all occupational diseases or that the word "injury" includes any type of occupational disease; and the "schedule" type of coverage, in which the laws list diseases for which compensation will be

paid in case of disability, or make special provisions as in the case of West Virginia, which compensates only for disability in cases of silicosis. It is interesting to note that 27 states as named, have made no provisions for the coverage of compensation for disability due to occupational diseases, and further, that four additional (or 31) states as named, make no provision for coverage of dust diseases.

It is of particular interest to those who are professionally concerned with the administration of compensation cases, what kinds of occupational diseases have been specifically named in the various schedules. Table XIX shows a summary of the occupational diseases scheduled, listing the diseases that are specifically covered, as of 1938.

It was of interest to know what diseases were common to all of the 10 existing schedules as shown. It was furthermore advisable to show what diseases are common to most of the existing schedules as listed under heading two, and finally, it was thought desirable to show the diseases least common to the existing schedules. With reference to the latter, there are some instances where a dseases is named by only one state—for example, African boxwood poisoning is named by the schedule of Minnesota only—anthraco-silicosis is found only in the provisions of Pennsylvania—and carbon dioxide poisoning is noted only in the Ohio statute.

Table XIX

SUMMARY OF OCCUPATIONAL DISEASES SCHEDULES
DISEASES SPECIFICALLY COVERED
1938

I. DISEASES COMMON TO 10 EXISTING SCHEDULES: Anthrax — Arsenic poisoning — Benzol poisoning — Caisson disease — Chrome poisoning (or ulceration) — Lead poisoning — Mercury poisoning — Phosphorus poisoning.

II. DISEASES COMMON TO MOST EXISTING SCHEDULES: Bursitis — Skin cancer or ulceration — Carbon disulphide poisoning — Radium poisoning (including radioactive substances) — Wood alcohol poisoning — Zinc poisoning.

III. DISEASES LEAST COMMON TO EXISTING SCHEDULES: Abrasions — African boxwood poisoning — Asbestosis — Anthraco-silicosis — Bone felon — Brass poisoning — Carbon dioxide poisoning — Dermatitis venenata — Formaldehyde poisoning — Frost bite — Hernia — Hydrogen sulphide poisoning — Infection or inflammation of skin — Manganese poisoning — Methyl chloride poisoning — Naphtha poisoning — Nickel carbonyl poisoning — Nitrous fume poisoning — Phthisis (stone workers' or grinders') — Pneumoconiosis — Potassium cyanide poisoning — Sulphur dioxide poisoning — Tenosynovitis — Volatile halogenated hydrocarbon poisoning — X-ray injuries.

It seems of importance that some method of standardizing a list of occupational diseases should be devised. Such a list has already been suggested (as referred to in the analysis of 1937) as being published in the Report of the Section on Insurance Law, dated February 16, 1937, and as suggested by the American Bar Association.

It would naturally be expected that in states where there is so-called "blanket" coverage, any one of the diseases named in Table XIX, if causing disability, might be compensable. Certainly those conditions named in this list have gained some recognition as being characteristic occupational diseases, although it must be said that there is considerable disagreement on this point, in reference to some.

Perhaps the best procedure at present is to follow the suggestions of the American Bar Association in reference to deciding what an accepted list of occupational diseases should be, as far as statutory coverage is concerned. The use of such a list, however, always must be governed by the conditions pertaining to any one case, especially with due regard for a cause-and-effect relationship between the injurious substances and conditions in the working environment as they *actually* (not potentially) exist, and the effect on exposed employees as shown by properly interpreted examination findings.

Conclusions

IF WE assume that the foregoing experiences as reported in this paper can be accepted as typical, we may conclude that:

1. There has been a material increase in the number of compensated cases of disability due to occupational diseases, as reflected in the experiences of New York and Wisconsin.

2. Occupational skin affections are by far the most frequent conditions encountered as causes of disease claims, particularly in states with extensive experience, such as New York, Ohio, and Connecticut. Exceptions to this were found in Wisconsin and Illinois, where silicosis was the leading occupational disease, as to severity in the former, and lead poisoning and silicosis were the most frequently encountered claims in the latter.

3. A closer analysis of the spread of dermatitis claims throughout various industries and occupations discloses the fact that there is a fairly even distribution among many different types of industries. This is probably due to the fact that the chief assignable causes are exposure to soap and water, and various dyes or dye products, which are found in many kinds of manufacturing.

4. The principal causes of denial of claims in one state (New York) were "no lost time"—"not over 7 days disability"—"not covered by law"—and "failure to prosecute."

5. That emphasis is being placed upon "all lung conditions" is shown by the fact that such a classification occupies a principal place among the causes for filing claims, among the groups in which claims were actually closed, and also that this was the principal cause for action in higher court decisions for 1936 and 1937.

6. The fact that no cases of certain types of occupational diseases have been reported, corresponding to certain schedule provisions, even for considerable periods of time (Ohio), is significant for that state only. The factors influencing such data are highly variable in the different states and must be considered for each state and in each instance.

7. As revealed by experience in one state (Connecticut), there is considerable discrepancy between the cases reported by physicians and the cases compensated but not so reported. Legislative provisions to cope with this situation have been set up in Connecticut.

8. Many people look upon silicosis as an unimportant disease because of its relative infrequency—although it is not rare, it is not common. However, figures relative to the cost of compensation for silicosis cases closed in the State of Wisconsin for 1936 revealed definite evidence to the contrary; although "noxious dusts," with 71 cases, aggregated only 6.6% of the total number of cases of occupational disease disability indemnified during 1936, the payment of compensation for these cases aggregated $119,737, or 55.4% of the total indemnity paid for the entire group of occupational diseases as noted. Furthermore, silicosis when compared with chief causes of injury, stood second highest in average compensation costs among the fatal cases, first among the permanent and total disability cases, and second among the relative permanent partial disability cases. We, therefore, conclude upon this evidence that the severity of silicosis is the highest of any occupational disease, of which we have adequate records at the present time. Moreover, in comparing the 1936 experience with that of the decade of 1920-1929 inclusive, the average compensation cost for all dust cases increased more than five-fold, while the total number of cases has increased over three-fold.

9. Court decisions are essentially legal interpretations of statutory provisions, and in some states include opinions regarding factual evidence. Generally speaking, such decisions reflect intelligence and mature consideration. Of obvious necessity, the character of court decisions is wholly dependent upon the occurrence of appeals from lower bodies. During 1936 and 1937, however, one is impressed by the number of decisions relative to silicosis cases. There was also definite emphasis on lung conditions in general.

10. In reference to kinds of statutory coverage at the present time, eight states make provision for "blanket" coverage; 13 states for "schedule" coverage and 27 states have no provisions for the coverage of disability resulting from occupational diseases—an additional four also making no provision for coverage of dust diseases, totaling 31 in all which do not provide for the coverage of disability resulting from dust diseases.

11. The trend of legislation is toward legal provisions for the reporting of occupational diseases; more accurate statutory definitions of the term "occupational disease;" the adoption of a schedule system for compensation of disability; and the appointment of special commissions for the investigation and study of methods of reducing or controlling hazards.

12. Present schedule systems of coverage show only partially what diseases should be included in a list of typical occupational diseases. There is evidence that in some states certain scheduled diseases do not give rise to claims. For example, in one state (Ohio) no cases have been reported on certain scheduled types of diseases during an entire decade. Perhaps the best procedure at present is to follow the 1937 suggestions of the American Bar Association.

13. Finally, the *severity* of silicosis (the direct cause of its high compensation cost) causes this disease to continue to be one of the most serious problems of the medicolegal phases of occupational diseases, and presents the most action-compelling reasons for the universal study and application of control measures.

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Traumatic Surgery Institute

—Proceedings of Meeting of April 29, 1938, at St. Luke's Hospital, Chicago—

THE following scheduled program was given with certain changes as hereafter indicated:

"Demonstration of Pathological Material," by DR. EDWIN W. HIRSCH.

"Treatment of Fractures of the Patella," by DR. JAMES J. CALLAHAN. (Lantern slides and film.)

"Peripheral Nerve Surgery," by DR. C. R. G. FORRESTER. (Lantern slides and film.)

"Traumatic Rupture of Intervertebral Discs," by DR. C. N. PEASE.

"Treatment of Fractures of Lower Leg," by DR. THEODORE L. HANSEN.

"Reconstruction Surgery of Foot," by DR. ROBERT O. RITTER.

"Bursitis and its Treatment," by DR. E. C. HOLMBLAD.

"Operative Treatment of Fractures of Neck of Femur," by DR. W. R. CUBBINS.

"Tuberculosis of Acetabulum; Infection Arthritis of Hip," by DR. EDWIN W. RYERSON. (Case reports and lantern slides.)

"Spot-Welding Injury of Hand with Infection," by DR. ROBERT L. KERRIGAN.

"Atrophic Charcot's Disease of Hip," by DR. ARTHUR H. CONLEY.

The evening meeting, at the Palmer House, had the following presentations:

"Circulatory Changes of Bones," by DR. FREMONT A. CHANDLER.

"Manipulative Surgery, with Special Reference to the Back and Pelvis," by DR. PHILIP LEWIN (By Invitation).

Discussion by DR. EDWIN W. RYERSON.

Morning Session

DR. EDWIN W. HIRSCH presented a demonstration of interesting pathological material, including various specimens and microscopic slides of tissue. The first case was relative to a kidney showing contraction and also hyaline degeneration —the heart showed sclerotic hypertrophy and myocarditis — the aorta was sclerotic — the liver showed chronic passive hyperemia and the lungs emphysema. The second case was one of tumor of the kidney in which the histological slides showed squamous cells arranged in mosaics. There was also demonstrated a case of meningioma on the right side of the middle cranial fossa; both gross and microscopic specimens were shown of this case. In conclusion, DR. HIRSCH presented a summary of the autopsy experience of the hospital for the current month.

DR. WILL LYON demonstrated a very interesting operative case, while the audience was awaiting the arrival of several of the scheduled essayists. The patient had suffered an accident due to the passage of a rod through the thigh, just missing the femur; the hamstring vessels and the sciatic nerve were involved. There was a good arterial supply, but no nerve supply. The wound had healed well and it was a question of whether one should graft the sciatic nerve. This did not seem practical, so, the man having recovered with good extensor action, it was decided to wait and see what further could be done. In the meantime he developed an edema and now he has a pathological ankle joint with central necrosis, such as occurs in syringomyelia and tabes. It will be necessary to amputate the leg and the question of point of amputation is important. It was DR. LYONS' belief that in this instance one should leave the lower end of the femur for weight-bearing and save the knee-joint and patella, since the proper fitting of an artificial leg and early walking can be expedited by the use of the amount of structures left by this operation.

DR. KELLOGG SPEED discussed DR. LYON'S presentation as did also DRS. RYERSON and HALL. The question of ankylosing the joint was discussed but one is reminded that the question of nerve and blood supply is one of importance.

DR. ROBERT L. KERRIGAN presented an unusual experience with reference to "Spot-welding Injury of the Hand with Infection." The methods of spot-welding as used expose the thumb, the web of the forefinger and themar eminence, as well as a portion of the palm. The sparks can pierce gloves, as happened in the present case, and there is a definite explosion of metal. The lung effects were also discussed in reference to other cases, and an exhibit of goggles used in such welding was passed around the audience, showing the pitting on the goggles. In this instance, the infection was cultured and the cause shown to be a hemolytic streptococcus. In reference to the explosion of metal in spot-welding, it is not known exactly what causes this difficulty, but it is known that there was no such difficulty arising with the older and slower welding machines. It is, therefore, believed that it is a question of speed.

DR. JAMES J. CALLAHAN, in discussing "Treatment of Fractures of the Patella," stated that there were about 600 cases in the series on which he was reporting. Such fractures are rather frequent and may be caused by either indirect or direct violence. In this series, 12 cases were compound in type and 375 cases were transverse. The remainder of the fractures were comminuted. Results obtained showed about 65% bony union, which is really a high percentage ordinarily. X-ray demonstrations showing the period of union varying from nine months to three years were given. There were 90% of the cases which obtained full function. The treatment may be divided into conservative and operative methods. DR. CALLAHAN does not use strapping because the moleskin cuts into the tissues and handicaps subsequent surgery. If the patient is able to extend the leg and there is no separation, the patient is put flat on the back and a plaster posterior mold applied. It should be stated that wiring is not adaptable to all fractures of the patella. Kangaroo tendon requires three knots to prevent slipping. The knots may produce thrombosis and a slipping of the suture subsequently. Fascia lata makes an excellent suture but produces scars. Braided silk has been found to be of greatest efficiency. The incision of the operation is important, and a long incision is made over the patella after which the fragments are brought into apposition with the retractor. The upper fragment is raised and the contents in this area expressed, to avoid adhesions. Then a circlage of braided silk is put in; the capsule is sewed with catgut; the posterior cast is applied, remaining in place for 10 to 14 days, after which the patient becomes ambulatory. A series of x-rays were shown of the results before and after treatment, and a motion picture showing the operative technique was demonstrated.

In the discussion, it was noted that the essayist is not in favor of removal of all or a part of the patella because it takes longer for return of even partial function. DR. RYERSON mentioned the use of silk many years ago and stated that the former trouble arising from its use was due to lack of complete asepsis. Since that time, this has been overcome by boiling the silk in a dilute solution of mercuric chloride (3: 5000) and then coating with a paraffin of low boiling point. After 20 years a case has been shown in which there was no infection, there being an opportunity to cut in on the former operation; these strands of braided silk appeared to be just like tendons in such cases.

Further discussion by DR. SPEED brought out the fact that it is important to consider the suturing of ligaments and fascia. It is also important to remember that bony union may not take place for a year in these fractures.

DR. C. R. G. FORRESTER, in discussing the subject "Peripheral Nerve Surgery," stated that peripheral nerve injuries have become more and more important. The war experience taught the possibility of suturing three- to five-inch gaps by using flexion of the joints. The question of apposition of relative axis cylinders was for a long time in doubt, but it has been confirmed by pathological sections. In suturing the sheaths, it was found that silk produced tumefaction and fibrosis. When 000 catgut is used, one must be careful to get all of the axis cylinders enclosed which if left out will proliferate and produce casusalgia. The sheath of the nerves must be traumatized as little as possible. The normal nerve must not be kinked but fascia should be interposed in making a gentle curve. It is of especial importance to do a good approximation. Many times in severe injuries, no thought of nerve injury is considered. Motion of the affected part should not be started earlier than 45 days because nerve regeneration will then be ruined. A motion picture showing the points in the technique of repair of fracture and nerve suture was given. In the discussion, two or more war experiences similar to those of the essayist were discussed.

DR .C. N. PEASE, in presenting the subject "Traumatic Rupture of Intervertebral Discs," stated that in the opinion of many the rupture of intervertebral discs is still a myth and that there are many who still believe that arthritis or congenital lesions are responsible for such changes. As a matter of fact, the rupture of intervertebral discs is one of the most common major injuries to the spine and is sometimes associated with fracture. Lantern slides illustrated the embryonic derivation of spinal structures and the normal anatomy of the spinal column including the structure of the cartilages (superior and inferior cartilage plates—the annulus fibrosis—and the nucleus pulposis). X-rays of various cases were demonstrated, showing the spurring of vertebral margins, closer fitting interarticular facets, and the narrowing of the interarticular foramina. These injuries were caused by falls, by being thrown to the floor while in automobiles, by sneezing, by falling off a platform, by falling into an automobile grease-pit, by tripping, by being struck by a moving automobile, and by diving into shallow water. In such instances, sometimes there are compression fractures of the body of the vertebrae.

In the discussion by DR. JOHN ELLIS, the work of Kimmel and Schmorl was cited. In 18% of a series of 2,000 cases there was a posterior prolapse of material without damage to the cord. Schmorl found only two cases of rupture into the body of the vertebra produced from single trauma. He later demonstrated that 35% of normals show such protrusion. Herniation into the vertebral body is not important unless it causes definite torsion of the spine, resulting in neurological symptoms. In further discussion by DR. WHITE, of Minneapolis, he brought out the use of lipiodol visulation of filling defects in the spinal canal.

Afternoon Session

DR. THEODORE L. HANSEN presented an x-ray demonstration of cases relative to "Treatment of Fractures of Lower Leg." Thorough cleaning of the wound is advised, closure of the wound, application of traction, and application of plaster direct to the skin. In one case demonstrated by x-ray, traction was applied by pins through the head of the tibia and os calcis—the cast was on for four to six weeks and then changed; the patient used an U-shaped walking iron in starting to walk. The essayist uses unpadded casts on all Potts' fractures, supplemented with the walking iron. Early walking prevents muscular atrophy, gives full range of joint motion, and in such cases there is no evidence of calcium absorption in the x-rays of the bones; further, physiotherapy in such instances is not ordinarily required.

DR. ROBERT O. RITTER demonstrated cases with regard to "Reconstruction Surgery of the Foot." The first case was double foot drop after poliomyelitis. The operation done was an arthrodesis of ordinary triple type. Such an operation prevents the twisting of the foot which is so often noticed in suspension operations. Similar operation was done on the other foot with transplantation of the anterior tibial tendon into the solid bony anchorage. It is also possible to get good results by transplating the tendon of the extensor longus hallucis.

In participating in the discussion, DR. S. PERRY ROGERS showed lantern slides of a case of a girl with a marked equinus. Patient must be 12 years of age or older to be successful with arthrodesis. DR. ROGERS used in this instance a modification of Campbell's bone block operation, known as Wagner's modification.

In the discussions of DR. HANSEN'S and DR. RITTER'S papers, there was a question of whether weight was put on too soon with the use of pins. When the fracture or wound is closer to the middle than to the ends of the bones, it may be advisable to use pins in the lower tibia. Shoes are much better than slippers. If suppuration occurs, a hole can be immediately cut in the cast. The use of the inverted Thomas splint for support is of some advantage and will give easy access to dressings. There has been no trouble arising from the use of casts directly on the skin. There have been instances of infection around the angle which did not affect the bone by way of the pins.

DR. S. PERRY ROGERS briefly gave the important points on tendon suture, as follows: (1) repair at once; (2) use non-absorbable silk sutures; (3) this is a major operation but a good technique can be done with a local anesthesia; (4) splint should be left on for 10 days to two weeks; (5) early active motion is advisable.

DR. E. C. HOLMBLAD spoke on "Bursitis and its Treatment." An important point to keep in mind is that one must decide what the etiology is, which will indicate proper treatment. There is

a good discussion of the subject in Christopher's "Minor Surgery." The most involved areas are the elbow, shoulder, and knee. A good history should be obtained with regard to a definite injury at a known time and an association of events and effects. There may have been direct contusions, causing hemorrhage or the extravasation of fluid. One must be careful to rule out old changes. Aspiration of fluid may be done, but is not practiced as much as formerly. One must be careful of tension of the pressure pad or bandage. Dr. Holmblad uses sponge rubber, a sample of which he demonstrated. Ace bandages are practical because they are elastic. It has been found in the experience of the essayist that the best after-therapy is long wave diathermy for acute types of bursitis. Suppuration should be treated as in any wound. In shoulder cases, one must differentiate tears of the supraspinatus tendon; for this the arm-over-head test is important, the tendon tear showing a jerk on lowering of the arms. One sees subdeltoid bursitis associated with abscessed teeth in about 75% of cases. It is, therefore, important to get x-rays of the teeth. One should also look for infection in the tonsils and prostate. In the use of long wave diathermy, one uses a piece of block tin as the electrode, and one must watch the case carefully to avoid burns. Motion is important within tolerance of the patient, except in the presence of suppuration. Pure gum rubber is used for direct compression over the knee joints and no trouble has been experienced with skin reactions. This rubber is available in three grades of texture and has lots of other uses.

In the discussion by Drs. Cubbins and Kerrigan, points about anti-rheumatic treatment and the rapid regeneration of bursa were made. There was also mention of the use of a sclerosing solution by Dr. Kerrigan.

DR. W. R. CUBBINS spoke on the subject of "Operative Treatment of Fractures of Neck of Femur." Typical procedures were well demonstrated by a motion picture film in color. In severe types of compound fractures, one must first cut away the dead muscular tissue, washing the wound with a sterile salt solution. If dirt has been ground into the bone, this must also be cut away. The wound is then closed. A pin is inserted through the heel and traction is exerted. Dr. Cubbins also discussed injuries due to crushing of the chest. There are some peculiar ideas as to disability in fracture of the sternum. It is common to have extensive emphysema, but he has not seen a single case of phlegmon. In crushing injuries of the chest, the chest sinks as the abdomen rises in breathing. It has been found effective to use plenty of morphine to slow the breathing; the chest cannot be strapped, for the patient will die. In the discussion of hand wounds, it has been found effective to suture loosely. As soon as the dressings become dry continuously, one can proceed with further steps. It is inadvisable to use any cleansing agent or to drain the wound. We should always use tetanus and gas antitoxin. In gas cases, x-ray treatment alone does not give satisfactory results; careful surgery is needed. Potassium permanganate and hydrogen peroxide have been found effective.

DR. EDWIN W. RYERSON gave case reports and lantern slides on "Tuberculosis of Acetabulum; Ineffective Arthritis of the Hip." We see fewer cases of tuberculosis of the joints in children at the present time, since we have had proper milk control. In a case of a boy of five years of age in 1923, there was definite evidence of tuberculosis of the acetabulum. He was treated by a long splint. In 1924 the splint was still kept on; in 1925 a groin abscess occurred. In 1927 there was a fusion operation done outside the joint and a splint was put upon the femur. In January, 1938, the last check-up made, there was a good result shown. In another case, a woman aged 40, who had had a tuberculosis of the acetabulum 20 years ago, good results were shown over that period. In a third case in a woman of 20 there was a combined intra- and extra-articular fusion done. In this instance, the patient got along very well, although there was a stiff hip joint. In still another case of a woman who had had tuberculosis of the hip as a child, the process healed without an operation. Later on, however, she had tuberculosis of the spine and a fusion operation was necessary. Several cases were mentioned in which there had been no operation and there was recovery, including a fractured neck of the femur in a tabetic, which healed without operation. An interesting case was that of a boy of five years who had cervical abscesses, pain in the arms and legs, in 1934. The right hip was put in Buck's extension; complications ensued and it was necessary to give a transfusion; there was limited motion in the left hip, so he was put in a hip splint and kept at rest and recovered in 1936 after walking on crutches with a traction splint.

DR. ARTHUR H. CONLEY presented five cases of "Atrophic Charcot's Disease of the Hip." Dr. Conley emphasized that the physician should always have in mind in injury cases, the possibility of a luetic infection. Especially important is the appearance of the Argyll-Robertson pupil with loss of reflexes; girdle pains or a loss of testicular reflexes; ataxia. It is important to find out about a history of chancre. All of these things point the way toward the necessity of having a blood test. Lantern slides were demonstrated to show the important points in the five cases reported upon. In the first case, the head of the femur had been removed but there was no hypertrophy of any type. The second case showed marked mobility of both hips with no pain. The third case showed discomfort of the right hip without any unusual findings. In the fourth case, the spinal fluid was positive and the Wassermann was positive; there were girdle pains and there was marked hypertrophy of bone. The bad features about such cases are the history of injury and many times the diagnosis of an old fracture

of the neck. These cases should be examined very carefully where injury is claimed.

In the discussion of DR. CONLEY'S paper, several very interesting experiences were detailed, particularly with reference to blaming an injury for the joint changes which are frequently ascribable to a luetic infection. DR. HAMMOND thought that more detailed histories should be taken and that where indicated, Wassermann reactions should always be done. In closing, DR. CONLEY discussed briefly the medicolegal aspects of such cases, recommending that the patient be put in the hospital for five or six weeks' observation and that a complete physical examination at the first opportunity may be the answer to this perplexing problem.

Central States Society

—Proceedings of Nineteenth Annual Meeting, Springfield, Illinois, May 17, 1938—

THE Nineteenth Annual Meeting of the Central States Society of Industrial Medicine and Surgery was held in conjunction with the Illinois State Medical Society Meeting at Springfield, Illinois, on May 17, 1938. The following program was presented:

"Medicolegal Trends in Occupational Diseases" (Lantern Slides), by DR. C. O. SAPPINGTON.

"Recognition of Early Tuberculosis in Industry," by DR. JAMES A. BRITTON.

"Industrial Solvents," by DR. WM. D. MCNALLY.

"Skin Affection in Industry, with Special Reference to the Value of the Patch Test," by DR. CLEVELAND J. WHITE.

"Diagnosis of Lesions of the Spine Producing Abdominal Pain," by DR. EDWARD L. COMPERE.

"Traumatic Lesions of the Male Urethra," by DR. HARRY CULVER.

"Subcutaneous Injuries of the Abdomen," by DR. FREDERICK CHRISTOPHER, Evanston.

"Injuries of the Right Upper Abdominal Quadrant," by DR. PHILIP H. KREUSCHER.

"Spleen Injuries," by DR. CHESTER C. GUY.

THE morning session pertained to industrial medical subjects and was presided over by DR. FREDERICK W. SLOBE, the President of the Central States Society. In the afternoon, the session was a joint one with the Surgical Section of the Illinois State Medical Society and both DR. SLOBE and DR. SUMNER L. KOCH, Chairman of the Section on Surgery, Illinois State Medical Society, presided.

DR. C. O. SAPPINGTON presented statistical material on lantern slides relative to occupation disease experiences during the years 1936 and 1937. The full text of DR. SAPPINGTON'S presentation, "Medicolegal Trends in Occupational Diseases," appears on pages 331-337 herein. He concludes that the trend of legislation is toward legal provisions for the reporting of occupational diseases; more accurate statutory definitions for the term "occupational disease;" the adoption of the schedule system for compensation of disability; and the appointment of special commissions for the investigation and study of occupational disease problems, especially relating to the methods of reducing or controlling hazards.

DR. JAMES A. BRITTON discussed "Recognition of Early Pulmonary Tuberculosis in Industry." In Illinois the death rate from tuberculosis has been reduced in the last half century from 250 or more per hundred thousand to less than 60 per hundred thousand. Figures were quoted showing that there has been considerable reduction in mortality in various large cities. Recent studies made of large industrial groups show that the death rate from pulmonary tuberculosis during the year 1937 was 14 per hundred thousand and that the incidence during that same period of known cases of pulmonary tuberculosis was 80 per hundred thousand. (DR. BRITTON'S paper appears in full on pages 303-307 herein.)

DR. WM. D. MCNALLY spoke on the important subject of "Industrial Solvents." There has been a great increase in the number and amounts of solvents manufactured at the present time over what existed formerly. A knowledge of the boiling point and the formula of any given solvent provides important evidence as to its toxicity. All the volatile chlorinated hydrocarbons are to be considered poisonous. Solvents exert acute and chronic poisonous effects, especially on the liver, nervous system and blood-forming organs. Closed systems are best for degreasing operations. Proper equipment should be used to avoid skin contact. Since the classic work of Dr. Casey Wood, of Chicago, in 1904, when he first called attention to the toxic effects of wood alcohol on the eye, a great deal of attention has been given to wood alcohol and it has been discovered that it is poisonous by any route of entrance to the body. Air exposures should contain less than one part per million. With reference to the various distillates of the benzine group, Hayhurst's important work was mentioned. (The full text of DR. MCNALLY'S presentation is published on pages 295-303 in this issue.)

DR. CLEVELAND J. WHITE reviewed 102 recent papers and also cited his own experiences in a paper "Skin Affections in Industry, with Special Reference to the Patch Test." In discussing this subject we are concerned with external irritants almost exclusively, and must rule out instances of previous fungus disease. The differential diagnosis brings in the whole gamut of the dermatidides. The two great classifications can be named as characteristic eruptions and eczematoid dermatitis. The use of the patch test has greatly increased in frequency and is in essence a miniature reproduction of the reaction. Eleven states now compensate for disability arising from industrial dermatitis. Important contributions were made by Markley in 1920 and Sulzberger in 1931, while Ingram did a great deal of work in fur in-

vestigations. While the patch test is important, other factors which are not to be disregarded are impossible of reproduction by the patch test. However, the patch test is of great diagnostic help and its use should be continued. Undoubtedly it gives a sensitization index. Wise and Sulzberger call attention to the necessity of elmination of certain kinds of substances and the substitution of other compounds for these. Forester has emphasized the importance of non-occupational exposures in reference to diagnosis and states that the patch test is valuable in differential diagnosis, although it has certain limitations. In a series of 589 industrial cases Downey recognized that the patch test had valuable points, but also stated that certain conditions cannot be reproduced. In conclusion, Dr. White stated that industrial dermatitis is exceedingly common and it is usually the eczematoid type which prevails. A very important point is the etiology together with clinical tests, so that one may know that there is an associated relationship.

DR. FREDERICK CHRISTOPHER, of Evanston, discussed the subject "Subcutaneous Injuries of the Abdomen." Various types of force causing abdominal injuries were mentioned and classified. Case histories of various kinds of abdominal injuries were given, including liver rupture, spleen rupture, and rupture of the jejunum. Less frequently, the kidney is injured and the bladder may be injured if distended. The small bowel is also most susceptible to injury. A series of x-rays demonstrated various types of cases, including one on pneumoperitoneum.

DR. EDWARD L. COMPERE read the first paper in the joint meeting with the Section on Surgery on the subject "Diagnosis of Lesions of the Spine Producing Abdominal Pain." He first stated that his subject should be changed to symptom complexes of abdominal visceral pain. Lantern slides demonstrated among other things a large table of the causes of back pain. In citing case histories, x-rays of the dorsal spine were shown in which narrowing of the intervertebral discs and lipping had occurred. In one case there had been no relief from a brace and physiotherapy. A gastro-intestinal x-ray showed the occurrence of a duodenal ulcer. In another case, a girl of 21 had pain in the region of the dorsal spine for 11 months; after investigation a chest film showed a bronchiectasis and also other procedures showed a double infection of the maxillary sinuses. It is important to remember that conversely pathology may be located in the spine and pain referred to the abdominal viscera. Diagrams were shown demonstrating according to Pottenger's outline the mechanism of radiculitis. In one instance, a passenger in an automobile was thrown off the seat and on to the floor; there was no x-ray of the spine taken; there was a distended abdomen but at operation there were no pathological findings; later on a compression fracture of the spine was found to have caused the paralytic ileus. In another instance, the patient was injured in an automobile collision; the appendix and the ovaries were removed; later on a diagnosis of cyst of the cord in the dorsal and lumbar vertebra was confirmed. In another case, the patient complained of inguinal pain; in a later study a complete collapse of the second lumbar vertebra was found, due to a benign giant cell tumor. The obvious point to keep in mind is to be certain about the source of the pain rather than to blame the referred locus of the pain. Only this can form a rational basis for proper treatment.

DR. HARRY CULVER spoke on "Traumatic Lesions of the Male Urethra." A lantern slide demonstration was given showing the sectional anatomy of the pelvis. Injuries to the urethra are usually the so-called "straddle" type. It is important to evacuate clots in perineal hemorrhages. Urethrotomy is another procedure which is usually effective; sometimes it is necessary to do a cystomy and retrogade catheterization. Local sepsis is often a difficult problem where early surgery is not done. A good procedure to hold the urethra in line is accomplished by passing a sound both above and below and then putting through a catheter. The perineal approach is recommended by some operators. Important points to keep in mind are the combating of urinary infection and the prevention of strictures.

DR. PHILIP H. KREUSCHER presented a paper on "Injuries of the Right Upper Abdominal Quadrant." First a review of the anatomy of this region was considered. In describing cases of rupture of the liver, several investigators have reported a mortality of 40% in various series. The usual procedure is to pack with gauze and drain. Various injuries to the kidney were mentioned and classified. One case of rupture of the stomach was described. Only six cases are reported in the literature relative to injury to the pancreas; it is important to keep in mind that extensive hemorrhage may occur after only slight injury to this organ. In summarizing his paper, DR. KREUSCHER stated that early operation was necessary in perforated wounds of the abdomen; the patient should have thorough blood, urine, and x-ray examinations, and repeated readings of the blood pressure, which should be raised for exploratory laparotomy. The cardinal rules are to avoid haste—take a careful and detailed case history—make a painstaking differential diagnosis—and finally, an operation by a competent and skilled surgeon.

DR. CHESTER C. GUY completed the program by a discussion on "Spleen Injuries." Such injuries average one in 3,000 injured patients. Considerable violence is ordinarily exerted, and the injury is usually characterized by marked hemorrhage and splenectomy is quite ordinarily necessary. Gauze packing is not dependable, but splenectomy is really indicated. Transfusion is important in primary hemorrhages, and secondary hemorrhage should not be overlooked.

Occupational Diseases : Industrial Medicine

—Twenty-Third Annual Meeting of the American Association of Industrial Physicians and Surgeons, and Second Annual Midwest Conference on Occupational Diseases—
—Official Programs—

FOLLOWING are the Official Programs for the Twenty-Third Annual Meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS, and the Second Annual MIDWEST CONFERENCE ON OCCUPATIONAL DISEASES, at the Palmer House, in Chicago, on June 6, 7, 8, and 9, 1938:

MONDAY MORNING
June 6, 1938

8:00 — 9:30 Registration (Fourth Floor)

C. O. SAPPINGTON, M.D., DR.P.H.
Chairman of the Session

9:30 OPENING OF MIDWEST CONFERENCE ON OCCUPATIONAL DISEASES, AND TWENTY-THIRD ANNUAL MEETING, AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS.

Address of Welcome.
HON. EDWARD J. KELLY,
Mayor, City of Chicago.

Response for the Association.
R. R. SAYERS, M.D., President.

9:35 **"The Engineer's Place in Industrial Hygiene."**
WARREN A. COOK, Supt. Engineering Dept., Zurich General Accident & Liability Insurance Company, Ltd., Chicago, Illinois.

Abstract:

THIS will show how the work of the engineer correlates with that of the physician in the prevention of occupational diseases and how the engineer can be most effective when he has available to him the advice and assistance of the industrial physician well versed in the medical aspects of industrial hygiene. Since one of the most difficult phases for the engineer in control of occupational diseases is the decision on the necessity, and the extent, of control measures, methods will be outlined whereby the engineer can more definitely guide his efforts in occupational disease control.

Discussion opened by H. H. SCHRENK, Ph.D., Chief Chemist, Health Division, U. S. Bureau of Mines, Pittsburgh.

10:15 **"The Insurance Company and the Control of Occupational Diseases."**
J. B. LAMENZO, Research Engineer, Hartford Accident & Indemnity Company, Hartford, Connecticut. (Lantern Slides.)

Discussion opened by A. S. GRAY, M.D., Director, Bureau of Occupational Diseases, State Health Dept., Hartford, Connecticut.

INTERMISSION

Inspection of Scientific and Commercial Exhibits.

11:00 **"The Physician's Responsibility in Industrial Hygiene."**
C. D. SELBY, M.D., Medical Consultant, General Motors Corporation, Detroit, Michigan. (Lantern Slides.)

Discussion opened by J. J. PRENDERGAST, M.D., Medical Director, Chrysler Corporation, Detroit, Michigan.

LUNCHEON

MONDAY AFTERNOON
June 6, 1938

C. O. SAPPINGTON, M.D., DR.P.H.
Chairman of the Session

1:30 **"Engineering Aspects of Problems in Industrial Epidemiology."**
J. J. BLOOMFIELD, P. A. Sanitary Engineer, Division of Industrial Hygiene, U. S. Public Health Service, Washington, D. C. (Lantern Slides.)

Discussion opened by JOHN M. HEPLER, Director, Bureau of Industrial Hygiene, Michigan Dept. of Health, Lansing, Michigan.

2:15 **"Plumbing as an Industrial Hygiene Problem."**
MAJOR JOEL I. CONNOLLY, Assistant to the President, Chicago Board of Health. (Illustrated by lantern slides and motion pictures.)

Abstract:

DRINKING water may be contaminated by cross-connections between city water and other water used for industrial or fire protection purposes. Several epidemics of disease among industrial workers from polluted water are cited. Also drinking water may be contaminated by inter-connections of the city water with tanks of poisonous chemicals, sewers, hydraulic presses, air conditioners and other fixtures. Typical cases are shown by stereopticon slides and motion pictures, and remedies described. Better still, means of prevention are outlined which have been found effective.

Discussion opened by J. H. CHIVERS, M.D., Medical Director, Crane Co., Chicago.

INTERMISSION

Inspection of Scientific and Commercial Exhibits.

3:30 **"Industrial Hygiene and the Nurse."**
JOANNA M. JOHNSON, R.N., Supervisor of Nurses, Employers Mutuals, Wausau, Wisconsin.

Discussion opened by GERTRUDE F. JAEGER, R.N., Swift & Company, Chicago.

4:00 — 6:00 FELLOWSHIP HOUR.

6:30 DINNER.

CAREY P. MCCORD, M.D.
Presiding Chairman.

"Air Conditioning in Industry."
DR. WILLIS H. CARRIER, Chairman of the Board, Carrier Corporation, Syracuse, New York.

Reports of Committees:
(1) American Association of Industrial Physicians and Surgeons
(2) American Society of Heating and Ventilating Engineers
on "Policies and Problems."

The Committee of American Association of Industrial Physicians and Surgeons is as follows:
E. C. HOLMBLAD, M.D., F.A.C.S., Chairman.
LOYAL A. SHOUDY, M.D., F.A.C.S.
EMERY R. HAYHURST, M.D.
CLARENCE D. SELBY, M.D., F.A.C.S.
GEORGE H. GEHRMANN, M.D., F.A.C.P.

The Committee of American Society of Heating and Ventilating Engineers includes:
JOHN P. AEBERLY, Chief of Division, Heating, Ventilation, and Industrial Sanitation Section, Chicago Board of Health.
WILLIS H. CARRIER, Carrier Corporation, Syracuse, N. Y.
PHILIP DRINKER, Professor Industrial Hygiene, Harvard University.
E. V. HILL, M.D., Editor, *Aerologist.*
JOHN HOWATT, Chief Engineer, Board of Education, City of Chicago.
A. C. WILLARD, President, University of Illinois.
B. M. WOODS, Professor of Engineering, San Francisco.

TUESDAY MORNING
June 7, 1938

WM. D. MCNALLY, M.D.
Chairman of the Session

8:00 — 9:00 **Visiting Exhibits**

9:00 **"Contact Dermatitis."**
MICHAEL EBERT, M.D., Associate Professor of Dermatology, Rush Medical College, Chicago.
Discussion opened by HERBERT RATTNER, M.D., Chicago.

9:30 **"Practical Problems Under Blanket Coverage for Occupational Diseases: A Review of the Illinois Claim Experience."**
CLARK BRIDGES, Casualty Mutual Insurance Company, Chicago.
Discussion opened by C. O. SAPPINGTON, M.D., Dr. P.H., Chicago.

10:00 — 10:45 **"Noise in Industry."**
WALLACE WATERFALL, Director of Research Engineering, The Celotex Company, Chicago.
Discussion opened by CAREY P. MCCORD, M.D., Detroit.

INTERMISSION
Inspection of Scientific and Commercial Exhibits.

11:15 — 12:00 **"Developments in Occupational Disease Legislation."**
OLIVER G. BROWNE, General Claims Attorney, New York Central System, New York City.

LUNCHEON
Inspection of Scientific and Commercial Exhibits.

TUESDAY AFTERNOON
June 7, 1938

ANDREW M. HARVEY, M.D., F.A.C.S.
Chairman of the Session

1:30 **"Women in Industry."**
M. H. KRONENBERG, M.D., Chief of Division and Industrial Medical Supervisor, State of Illinois Department of Public Health, Chicago.
Discussion opened by R. J. DEMOTTE, M.D.

2:00 — 2.45 **"Light, Vision, and Seeing."**
MATTHEW LUCKIESH, D. Sc., Director, and FRANK K. MOSS, Physicist, Lighting Research Laboratory, General Electric Company, Nela Park, Cleveland, Ohio.

INTERMISSION
Inspection of Scientific and Commercial Exhibits.

3:15 **"Tuberculosis in Industry."**
O. A. SANDER, M.D., Milwaukee, Wisconsin.
Discussion opened by JEROME R. HEAD, M.D., Chicago.

4:00 **"The Employability of the Silicotic."**
VOYTA WRABETZ, Chairman, Industrial Commission of Wisconsin, Madison, Wisconsin.

5:00 — 6:00 FELLOWSHIP HOUR.

TUESDAY EVENING
Open

WEDNESDAY MORNING
June 8, 1938

FREDERICK W. SLOBE, M.D., F.A.C.S.
Chairman of the Session

9:00 Opening of the Twenty-Third Annual Meeting of the
AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS

9:15 **President's Address.**
ROYD R. SAYERS, M.D.

10:00 **"Hazards of Night Driving."**
HART E. FISHER, M.D., F.A.C.S., Medical Director, Chicago Rapid Transit Co., Chicago.

Abstract:

A NIGHT-DRIVING hazard exists; 60% of all fatal motor vehicle accidents occur at night when only 25% of the daytime traffic is on the road. Therefore, the fatalities are greater in motor vehicle accidents and, like-

wise, injuries, when the exposure is only one-fourth of the traffic of daytime motor vehicle operation. Speed is the direct cause of night hazards, when the visibility of the driver is confined to the headlight beam of an average distance of about 200 feet ahead of the vehicle. At high speed it is impossible to retard the movement of the vehicle if a dangerous condition arises beyond the extent of the headlight beam.

Likewise, the fields of vision are narrowed to the ribbon of the headlight illumination and generally to the width of the pavement. On account of darkness there is comparatively little, if any, visibility at any angle beyond 30° of the fields.

The greatest hazard of night driving is the inability of the normal eye to adapt itself from glaring headlights to the dark adaptation following the passing of the glare.

The visual purple in the retina of the eye is greatly reduced in the intensity of the headlight glare; in many individuals the regeneration of this visual purple following glare adaptation is slow, and during this period the driver has difficulty in seeing—if not for a few seconds driving blindly. The driver's estimation powers as to depth, distance and motion are impaired.

Night blindness has been productive of a number of accidents, and generally the person having such an accident is innocent of that fact. Driving at night requires concentration of mind and effort, and the nervous stability of the average individual becomes upset. Likewise, mental and body fatigue are present more in night driving than in day-time driving.

The solution of night driving problems, with the reduction of the high mortality rates in night driving, can only be accomplished through the medium of education of the individual as to his physical limitations and the proper illumination of accident-peak areas on our streets and highways.

10:15 **"Factors Determining Reasonable and Equitable Temporary Total Disability Periods."**

PHILIP H. KREUSCHER, M. D., F.A.C.S., Chief Surgeon, Chicago Area, Carnegie-Illinois Steel Corporation; Former Medical Director, Industrial Commission of Illinois.

Discussion opened by VOLNEY S. CHENEY, M.D., F.A.C.S., Chicago.

10:45 INTERMISSION.

Inspection of Scientific and Commercial Exhibits.

11:00 **"The Occupational Disease Research Department of Northwestern University."**

FRED FITZ, M.D., Northwestern University Medical School.

Abstract:

UNIVERSITY recognition of the increasing prominence of occupational disease; organization of the department; general principles and purposes; research activities; case studies (hospital and out-patient); occupational surveys; relation to medical departments of cooperating industries; occupational disease undergraduate and graduate instruction; occupational disease library; annual symposium.

11:15 **"Syphilis in Industry."**

HAROLD A. VONACHEN, M.D., Medical Director, Caterpillar Tractor Company, Peoria, Illinois.

Discussion opened by ROYD R. SAYERS, M.D. and A. E. RUSSELL, M.D., U. S. Public Health Service, Washington, D. C.

12:00 LUNCHEON.

WEDNESDAY AFTERNOON
June 8, 1938

Program Presented by the Association for the Advancement of Industrial Medicine and Surgery

CASSIUS H. WATSON, M.D., F.A.C.S.
Chairman of the Session

1:30 **"Arthritis in Industry."**

JOHN DEPAUL CURRENCE, M.D., New York City.

Discussion opened by JOHN C. COULTER, M.D., F.A.C.S., Chicago.

2:15 **"Tuberculosis in Industry."**

GEORGE G. ORNSTEIN, M.D., F.A.C.P., New York City.

Abstract:

TUBERCULOSIS is discussed under the two main headings:

1. As an occupational disease.
2. The activation of pre-existing tuberculosis.

Inasmuch as tuberculosis is caused by the tubercle bacillus there are only a few instances in which the disease may be listed as an occupational disease:

1. The workers caring for the tuberculous patient—nurses, attendants, orderlies, kitchen, elevator men, etc., in a tuberculosis hospital.
2. Those persons subjected to silica dust (belief that silicosis produces susceptibility to tuberculosis). This belief has not been established to the satisfaction of all investigators.
3. The possibility of direct infection in those positions in which the worker converses with large numbers of people continuously day after day—food handlers, saleswomen, etc.

The above conditions are fully discussed.

In industry, tuberculosis is frequently discussed from the point of view of a reactivation of a previous existing disease by trauma or other causes capable of activating tuberculosis. Inasmuch as there is a pre-existing tuberculosis the responsibility of the insured to the employer will be discussed from the following points of view:

1. Competent, activating causal factors.
2. When does the responsibility of the insured cease.

Discussion opened by BENJAMIN GOLDBERG, M.D., F.A.C.P., Chicago.

3:00 INTERMISSION.

Inspection of Scientific and Commercial Exhibits.

3:30 **"Intra-Articular Damage."**

WILLIS W. LASHER, M.D., F.A.C.S., New York City.

Abstract:

PATIENTS suffering from intra-articular lesions have fixed ideas. Fractures of the long bones produce immediate disability. Joints function under adverse circumstances. Joint conditions must be satisfactorily explained to the patient in his own language. Recurrent effusions are of greatest importance diagnostically and therapeutically. We have found a "try-out" test of great value. Over-production of bone and not non-union, is a serious problem in all articular injuries. Arthritis dessicans is most generally recognized as a comparatively common condition. Injuries to the fibro-cartilagenous structures and the matter of aggravation of pre-existing arthritic conditions in relation to trauma will be touched upon.

Discussion opened by JOHN DEPAUL CURRENCE, M.D., New York City.

4:15 **"Lead Poisoning in Industry."**

IRVING GRAY, M.D., F.A.C.P., Brooklyn, New York.

Abstract:

THE physical findings and laboratory studies, including cardiovascular examinations will be discussed. The cases studied were all industrial in origin. The importance of a high phosphorous regime in the treatment of the acute toxic lead episode will be stressed. The necessity for occasional deleading in isolated instances, when medical legal problems are involved will be shown.

Discussion opened by E. L. BELNAP, M.D., F.A.C.P., Milwaukee, Wisconsin.

5:00 — 6:00 FELLOWSHIP HOUR.

6:30 **Annual Banquet.**

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS

WILLIAM A. SAWYER, M.D.

Presiding Chairman.

8:00 **Symposium.**

Employability and Job Placement from the Medical Point of View:

(a) **"Employability of Persons Presenting Disability or Disease."**

HARVEY G. ELLERD, Director of Personnel, Armour and Company, Chicago.

(b) **(To be announced.)**

Discussion opened by M. N. NEWQUIST, M.D., Chicago.

THURSDAY MORNING
June 9, 1938

Report of the Nominating Committee. Election.

Program Presented by Institute of Traumatic Surgery Chicago

CLARENCE D. SELBY, M.D., F.A.C.S.

Chairman of the Session

9:00 **"Discussion of Fat Embolism."**

CARLO S. SCUDERI, M.D., F.A.C.S., Chicago.

Abstract:

IN MY paper I will give a brief review of the high lights on the subject. This will be followed by both clinical and experimental observations. The newer aspects of the diagnosis of this disease will be presented. Detailed description of the technique of blood, urine, chest x-rays and surface tension will be given.

Discussion opened by WILLIAM R. CUBBINS, M.D., F.A.C.S., Chicago.

9:30 **"Trauma to Blood Vessels, and Their Sequelae."**

RAYMOND W. MCNEALY, M.D., F.A.C.S., Chicago.

Discussion opened by HARRY E. MOCK, M.D., F.A.C.S., Chicago.

10:00 **"Distinction Between Injuries of Spine, Spondylitis, and Spondylosis."**

JOHN D. ELLIS, M.D., F.A.C.S., Chicago.

Discussion opened by LOYAL A. SHOUDY, M.D., F.A.C.S., Bethlehem, Pennsylvania.

10:30 INTERMISSION.

Inspection of Scientific and Commercial Exhibits.

11:00 **"Pectin in the Treatment of Wounds."**

JAMES E. M. THOMSON, M.D., F.A.C.S., Lincoln, Nebraska.

Abstract:

MY INTEREST in Pectin was created through the discoveries of my friend, DR. CHARLES A. THOMKINS, of Detroit, who first used it advantageously in the treatment of infantile diarrhea. Bacteriological study of the bactericidal affect of Pectin led into the deduction that it varied inversely with the pathogenicity of the organism. For example, while Pectin kills streptococcus rather easily, staphylococcus are more resistant to its bactericidal affect. I have noted with interest these and other bacteriological studies by the Pectin Committee of the Lincoln General Hospital, and made some rather definite clinical observations with respect to a 2% aqueous solution of Pectin on infected wounds. *First*, that the wound treated with Pectin hasn't the usual fetid odor, but rather has a clean smell. *Second*, that the granulations are nonpurulent, healthy and bright in appearance. *Third*, although the Pectin seems to stimulate granulation growth the granulations are smooth and even with the skin, and it seems to discourage the unhealthy excess thick granulations so often seen. *Fourth*, healing time is markedly diminished over any other method. *Fifth*, in the healing instead of a piled-up scar there is a smooth epithelialized surface. *Sixth*, and lastly, we are of the impression that in the treatment of osteomyelitis, where there is sequestra formation, there is an earlier separation of the sequestra, which we believe in influenced by the use of Pectin.

Discussion opened by J. DANIEL WILLEMS, M.D., F.A.C.S., Chicago.

11:30 **"Metallic Fixation of Bones."**

WILL T. LYON, M.D., Chicago.

Abstract:

IN SPITE of improvements in skeletal traction and external reduction of fractures, a small percentage still requires internal fixation and some of these are best immobilized by a properly applied plate. A presentation will be made of experimental work being carried on in dogs, as well as clinical application in actual practice to illustrate the improvements suggested both in the mechanics of the application of the plates and their construction.

Discussion opened by JAMES J. CALLAHAN, M.D., F.A.C.S., Chicago.

12:00 LUNCHEON.

THURSDAY AFTERNOON
June 9, 1938
Chicago Regional Fracture Committee Symposium for AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS
Presented by the American College of Surgeons

KELLOGG SPEED, M.D., F.A.C.S.

Chairman of the Session

1:30 — 2:00 **"Fractures of the Carpal Navicular Bone."** (With Lantern Slides.)

Kellogg Speed, M.D., F.A.C.S.

2:00 — 2:30 **"Prevention vs. Cure in Deformity Following Fracture."**
Paul B. Magnuson, M.D., F.A.C.S.

2:30 — 3:00 **"March Foot."**
(With Lantern Slides.)
Dean L. Rider, M.D.

3:00 — 3:30 **"Treatment of Fractures of the Neck of the Femur by Priming."**
(With Lantern Slides.)
Willis J. Potts, M.D., F.A.C.S.

3:00 — 4:00 **"Treatment of Fractures of the Neck of the Femur."**
(With Moving Picture Demonstration.)
Hugh McKenna, M.D., F.A.C.S.

Collaborating Organizations

THE collaborating organizations now include: State of Illinois, Division of Industrial Hygiene;

Department of Public Health and Hygiene, University of Illinois College of Medicine;

Chicago Society of Industrial Medicine and Surgery;

Institute of Traumatic Surgery;

Board of Health, City of Chicago;

Occupational Diseases Research Department, Northwestern University Medical School;

Industrial Hygiene Unit, Wisconsin State Board of Health;

Chicago Medical Society;

National Safety Council;

Greater Chicago Safety Council;

Chicago Dermatological Society;

Department of Health, State of Michigan;

Bureau of Industrial Hygiene, Detroit Department of Health; and

Bureau of Occupational Diseases, Ohio State Department of Health.

Exhibitors

Booth 64

CLEVELAND J. WHITE, M.D.

THE exhibit of industrial dermatoses with special reference to the value and technic of patch tests will be under the auspices of the Chicago Dermatological Society. Charts will be shown of the various salient features of industrial dermatosis and the differential diagnosis. A number of photographs will be shown of the main features of occupational cutaneous syndromes. There will also be demonstrations of the technic of the application and interpretation of the patch test by various members of the society.

Booth 57

CHRYSLER CORPORATION

JOHN J. PENDERGAST, M.D., Medical Director, Chrysler Corporation, Detroit, Michigan.

Representative Historical and Contemporary Investigators of Occupational Diseases.

Names and faces make news and challenge interest. On this account, an exhibit will be made of all available photographs of representative historical and contemporary industrial hygienists and occupational disease investigators, and others.

In view of Industrial Medicine's increasing attention to occupational diseases, many physicians and others in attendance at this meeting will be interested in photographs of those who have made outstanding scientific contributions in the field of industrial diseases and industrial hygiene.

Booth 34

ORGANIC SILICON COMPOUNDS

ORGANO - SILICON Compounds, their Preparation, Purification and Identification, exhibited by the Bureau of Industrial Hygiene of the Detroit Department of Health. Demonstrated by William G. Fredrick, William N. Witheridge, and Herbert T. Walworth.

Owing to the widespread interest shown by industrial hygienists in the organo-silicon compounds just coming into industrial use, a working exhibit illustrating the principal phases of the preparation, purification, and identification of these compounds will be made. Not only will examples of the silico-esters and hydrocarbons be shown, but also the preparation, purification, identification and analysis of tetra-ethyl-silicane will be continuously demonstrated. The apparatus displayed will include a multiple-plate fractionating column for sharp separation of complex solvent mixtures, a precision boiling point apparatus, an Abbe refractometer and a Parr combustion bomb. Their operation and use will be illustrated and demonstrated.

Booth 17

HILGER PERRY JENKINS, UNIVERSITY OF CHICAGO, DEPARTMENT OF SURGERY, CHICAGO.

AN EXPERIMENTAL and clinical study of the decline in tensile strength and ultimate absorption of plain and chromic catgut is presented: a series of transparent tissue specimens showing the status of the catgut at varying periods of time after implantation; specimens of catgut removed from the tissues at varying periods of time which are mounted on cards with full description of the loss of strength which was observed for each size used; and by photomicrographs showing the reaction in the tissues to the catgut.

Booth 67

GENERAL MOTORS

IN THE General Motors exhibit, it is proposed to show by photographs and diagrams the measures used to control occupational diseases and the results obtained.

Booths 73 and 74

JAMES A. JACKSON, THE JACKSON CLINIC, MADISON WISCONSIN.

EXHIBIT of: (a) Transparencies demonstrating various types of injuries to the lower extremities, including special instruments and appliances used in their management.

Booth 45

PERIPHERAL CIRCULATION

FROM The Circulatory Group of St. Luke's Hospital, Chicago, by Carl A. Johnson, M.D. The purpose of the exhibit is to demonstrate the importance of the study of the circulation in certain diseases, with particular reference to those of traumatic origin.

The various instruments used to

make these studies will be demonstrated, particularly the digital plethysmograph with which most of the studies were made.

The results of studies of the peripheral circulation in various types of diseases will be presented by means of "transparencies," case reports, and short lantern-slide discussions. The case reports used will include normals, frost-bite, traumatic vascular occlusion, traumatic neuritis, lymphatic obstruction, multiple arterial occlusion, Buerger's disease, Raynaud's disease, and cases with muscular pain from anemia, generalized cardiovascular disease and faulty weight distribution.

Booth 60

MOCK, H. E., M.D.: SKULL FRACTURE EXHIBIT

TITLE: SKULL FRACTURE EXHIBIT: Exhibitors — HARRY E. MOCK and JOHN L. LINDQUIST — Surgical Department, Northwestern University School of Medicine, and St. Luke's Hospital.

The exhibit shows the need for improved management of skull fractures. It is an analysis of 3,000 collected cases and of 300 of the authors' own cases. Cases are grouped according to signs and symptoms. Another grouping based on the type of treatment. Analysis of indications and value of spinal puncture. Analysis of operative cases. Models showing skull and brain pathology, photographs, graphs, charts, and x-ray films.

Booth 36

NATIONAL SAFETY COUNCIL

THE NATIONAL SAFETY COUNCIL is an association of nearly 5,000 employers, insurance companies, associations, government departments and individuals. Its objects are to provide accident prevention service to its members and to promote the cause of safety everywhere. It gathers and tabulates information on the frequency and severity of accidents and on their causes. It investigates and reports methods of preventing accidents.

Some of the Council's publications, including magazines, Safe Practices Pamphlets, and posters are shown in the exhibit.

Booth 54

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

REPRODUCTIONS of serial chest roentgenograms of nine individuals having carcinoma of the lung are displayed. Eight of these have been proved histologically. Three additional suspicious cases are not included because of lack of confirming data. These cases were collected in 1937 from a group of about 40,000 employees in several different industries. The occupational histories reveal very little in common in the cases presented. Gross specimens of three of the cases are also to be on display.

Booth 15

LIBERTY MUTUAL

A SIMPLE exhibit on the Petrographic examination of dusts to determine the free percentage of silica in them.

Booth 27

OHIO DEPARMENT of HEALTH'S MOBILE LABORATORY

TO FACILITATE the study of silicosis, OHIO's Mobile X-Ray Laboratory was conceived in the summer of 1936. After months of planning and construction, it was put in operation in October, 1937. The unit consists of a special light-proof body mounted on a cab-over-engine truck with a wheel-base of 172 inches. The cab-over-engine truck was selected to give maximum space for the body with minimum wheel-base. The dimensions of the body are: Length 16 fee,t width 7 feet, 11 inches, and height 6 feet, 6 inches. A partition divides the body into two compartments. The rear compartment contains laboratory benches, cabinets, sink, and water tank.

The forward x-ray compartment contains tube stand, transformer, cassette changer, control panel, fluoroscope, lead-lined film box and ventilating appartus. The unit is shock-proof. Both flat and stereoscopic exposures are taken. Technique adopted is 100 milliamperes, 1/10th second at 72 inches with variable kilivoltage. The unit operates on 220 volts, 60 cycle current which is supplied at the plant. A special cable connects the truck with factory current.

Booth 4

MEDICOLEGAL TRENDS in OCCUPATIONAL DISEASES

C. O. SAPPINGTON, M.D., DR. P.H. Consulting Industrial Hygienist Chicago:

Occupational diseases present problems of increasing interest, especially relative to the medicolegal phases.

In this exhibit, material in the form of maps, diagrams, tabulations, and graphs will be presented, showing medicolegal trends in occupational disease problems.

Because the state laws vary considerably and have changed a great deal in the past 10 years, comparisons will be made showing advances; present types of coverage will be shown, especially relative to the rights of election, common law relations, and coverage for dust diseases. Coverage relative to other occupational diseases will be exhibited.

Compensation experience in various well developed industrial states, such as New York, Ohio, Wisconsin, Connecticut, Massachusetts, and Illinois will be shown in tabulations, comparing 1936 and 1937 experiences.

Since court decisions form the common law background, various tabulations of published court decisions during 1936 and 1937, according to disease alleged and geographical distribution, will be presented.

An outline of the suggestions of the American Bar Association, relative to legal provisions for occupational disease problems, will be shown.

Booths 6 and 7

NATIONAL INSTITUTE OF HEALTH, U. S. PUBLIC HEALTH SERVICE

A DISPLAY of the literature pertaining to industrial hygiene. There will be a central portion made up of panels with tabulated references from the current literature, divided into appropriate groups. Two other panels will deal with reference lists of textbooks and current periodicals that are useful to persons working in the field of industrial hygiene.

Booth 80

AMERICAN COLLEGE OF SURGEONS, INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY

THE exhibit consists of a symbolic diorama with drawings, charts and map illustrating different phases of medical service in industry. The annual per capita medical cost in industry, medical and compensation costs according to the size of the plant, and the percentage of distribution of the costs are shown by charts. Other graphs show the incidence of absenteeism from illness and injury in industry, the reduction in the injury frequency and severity rates.

Samples of physical examination record forms for use in industry will be available.

Commercial Exhibits

Booth 13

BROOKS APPLIANCE COMPANY

WE ARE displaying an extensive line of Ambulatory supplies, which you can advantageously use in your practice. Kindly leave your name at the booth so that we may send you our new catalogue describing these specialties in detail.

Peerless Injection Trusses. Post-operative and Surgical Supports. Varicose Vein and Varicose Ulcer Bandages. Vim Needles and Syringes. A proctoscrope especially designed for the Injection Treatment.

Booth 49

FAIRWAY LABORATORIES, INC.

BOOTH 49 in the exhibit hall, will bring an old friend of the industrial physicians to the convention. Mr. C. M. Schofield, President of FAIRWAY LABORATORIES, INC., of Belleville, Illinois, soon to move to their new factory in St. Louis, Missouri, will be present and demonstrate the greatly improved Fairway 1938 Molded Plastic Tablet Dispensers and other improved services. He also promises another of their informative booklets, entitled "Are Your Workers Worth Their Salt" which will be just off the press and presented to any industrial physician or safety director who may desire a copy. He looks forward to a pleasant visit with many medical directors of corporations with whom he has enjoyed correspondence on the interesting subject of "The Prevention of Heat Sickness" over the past nine years.

Booth 14

FARNSWORTH LABORATORIES

THE FARNSWORTH LABORATORIES are pioneers in the production of various types of solutions used for ambulatory methods of treatment. duction of various types of solutions used for ambulatory methods of treatment.

The Injection Treatment of Hernia is a popular subject, and one with which this company has long been identified.

A new Local Anesthetic having a prolonged action, extending over a period of days will be offered. Physicians who are interested in non-surgical forms of treatment should find a visit to this booth well worth while.

Booth 50

CAMERON SURGICAL SPECIALTY COMPANY

SEE THE new LEMPERT-LEMEE HEADLITE demonstrated in Booth No. 50 by CAMERON SURGICAL SPECIALTY COMPANY, Chicago. Latest developments in electrically lighted diagnostic and operating instruments for all parts of the body will also be shown. Of special interest will be the new inexpensive office model of a radio knife and other electro-surgical units for cutting, coagulating, desiccation and fulguration, ranging in size up to the "Hospital Model" which has sufficient power for the most radical and heavy surgery. Also see the CAMERON HEARTOMETER, the outstanding achievement in cardiac diagnosis.

Booth 78

DEPUY MANUFACTURING COMPANY

SPLINTS for you by DEPUY MANUFACTURING COMPANY will be shown in Booth No. 78 at the American Association of Industrial Physicians and Surgeons Meeting in Chicago. The finest quality of materials are used in constructing the different appliances. MR. W. D. BATES will be there to answer your questions. Your problem will have his serious consideration. Fracture Appliances from head to foot. Always something new. Everything in Vitallium. Come in and see us. You are welcome.

Booth 71

DEQUIN PHYSICIANS PRODUCTS COMPANY

AS MANUFACTURERS and distributors of pharmaceuticals of high therapeutic value, we are displaying ALPARENE for the Injection Treatment of Hernia and Hydrocele. ALPARENE is a product of painstaking animal research and clinical trial. ALPARENE is of suspensoid character, Non-Toxic, painless, and produces the mature, dense fibroblastic repair tissue so sought for in the ambulant treatment of Hernia. We invite you to visit our Booth No. 71.

Booth 66

THE DIONOL COMPANY

PLAIN DIONOL: Selected, processed solidified hydrocarbons. A pure white unguent which readily liquefies at body temperature to promote penetration to the entire treated area affording a healing, protective film. USES: Burns of all degree, skin redness or abrasion, cuts, boils, dermatoses, and skin con-

Floor Plan of the Exhibit Hall

ditioning. PODIZED DIONOL: Plain Dionol with 2% iodine oleate added in a non-irritating, non tissue staining form. Used by major industrial organizations for the primary application for general plant injuries, to promote quick healing, prevent infections or complications and afford protection. METHYLATED DIONOL: An analgesic rub for plant strains or muscular soreness. NASAL DIONOL: To check incipient head cold epidemics.

Booth 42

DOMORE CHAIR COMPANY, INC.

THE DOMORE CHAIR COMPANY will exhibit seating for the executive, the office personnel, and factory workers. DR. J. R. GARNER, of Atlanta, Medical Adviser of the DOMORE CHAIR COMPANY, will be in charge and will be glad to give information to those interested. DOMORE chairs are adjustable, so they can be made to fit the physical characteristics of the individual. They make it easy to sit correctly with comfort. The executive models provide proper support while at work, and through the application of special back construction, enable an executive to relax or take exercise when opportunity permits. DOMORE chairs are for the "Man Who Never Grows Old," and for the "Woman Who Wants to Stay Young."

Booth 53

HOLLAND-RANTOS COMPANY, INC.

THE HOLLAND-RANTOS COMPANY will exhibit its complete line of specialties: the Koromex diaphragm and Koromex vaginal jelly, the new H-R Emulsion Jelly, the Koromex diaphragm introducer and Kahma Powder method of contraception. Two surgical silk fabrics—Rantosilk and Hollandex—used extensively in hospitals for bedding and also made up into garments, such as surgeons' aprons, patients' throws, etc. The Rantos Fever Bag is designed for use as an accessory to various types of fever inducing apparatus. Its primary function is that of a heat retainer. The Powdex Vaginal Insufflator with the various cinquarsen and stovarsol formulae is used for the dry treatment of trichomonas vaginitis.

Booth 62

HORLICK'S MALTED MILK CORPORATION

THE HORLICK'S MALTED MILK CORPORATION, in Booth No. 62, will exhibit HORLICK'S, the Original Malted Milk, Powder and Tablet forms. They will distribute samples of the Tablets, as well as literature. Among the special uses of HORLICK'S MALTED MILK which will be featured are its advantages as an easily digested, nourishing food-drink, notably in nervous and digestive disorders, in gastric and duodenal ulcers, and wherever a food of ready assimilability is indicated. The Tablets offer a practical solution to the problem of between-meal feedings for ambulatory ulcer patients, as well as a convenient source of quickly available food-energy for the general factory and office worker.

Booths 81 and 82

EICHENLAUBS

EICHENLAUBS, Pittsburgh, Pennsylvania: Manufacturers of Wood Hospital Furniture for Deluxe Private Rooms, Solaria, Lounge and Dining Rooms: Nurses Groups and Special Equipment: Manufacturers Representatives for the NEW HAYNES SPRING. Consult our engineers on any difficult furnishing problems. No cost or obligation for this service.

Booth 75

LaMOTTE CHEMICAL PRODUCTS COMPANY

LAMOTTE BLOOD CHEMISTRY UNITS for the Physician will be exhibited in Booth No. 75. The apparatus will be in charge of chemists who will be only too glad to give full information concerning the operation of these simplified outfits for blood and urine analysis. The new outfit for determining Sulfanilamide in Blood and Urine will be on display. The approved KLINE SLIDE TEST for Syphilis will also be explained to any physician interested in this new test. Standard units for Blood Sugar, Blood Urea, Creatinine, Uric Acid and Calcium-Phosphorus will, of course, be shown. Full information concerning all of the tests will be cheerfully given, without any obligation whatever on the part of the visitors to this exhibit.

Booth 44

KEYSTONE VIEW COMPANY

CUMMINGHAM'S STEREOSCOPIC ATLAS OF ANATOMY: 324 stereoscopic photographs of prepared dissections of every part of the human body. A quick and ready reference presenting many possibilities for usefulness in emergencies as well as routine cases.

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1. The visually unskilled from those whose employment requires visual precision.

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Booth 58

J. B. LIPPINCOTT COMPANY

J. B. LIPPINCOTT COMPANY will display NEW books; BACON —Anus, Rectum and Sigmoid Colon; THOREK—Modern Surgical Technic; KRACKE—Diseases of the Blood and Atlas of Hematology; WILSON — Fractures; WOLF—Physician's Business . . . and New Editions of old favorites: MCBRIDE — Disability Evaluation; REHBERGER—Quick Reference Book of Medicine and Surgery; THOREK—Surgical Errors and Safeguards; and MEANS — Thyroid and Its Diseases. See also the NEW International Clinics edited by DR. GEORGE MORRIS PIERSOL.

Booth 32

S. M. A. CORPORATION

S. M. A. CORPORATION, manufacturers of products to meet specific nutritional needs, will exhibit the following:

SMco Carotene-in-Oil (Carotene is Pro-Vitamin A)—Vitamin A deficiency affects ability to adapt to light and darkness, and hence influences color differentiation and close visual application.

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THE physician who is served by the SUTLIFF & CASE pharmaceutical institution finds more than 1000 medicinal products from which to choose his weapons to fight disease and death.

THIO-CARA and THIOCYAN-TABS (Sutliff & Case pharmaceuticals) are preparations intended for judicious thiocyanate therapy in arterial hypertension. THIO-CARA is an aromatic liquid form of thiocyanate, while THIO-CYAN-TABS are specially coated tablets of calcium thiocyanate.

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age of stannic oxide and medicinal tin, used in furunculosis, carbuncles, styes, acne and other conditions of local infection of staphylococci origin.

Booth 79

PHILIP MORRIS & CO. LTD. INC.

PHILIP MORRIS & CO. Ltd., Inc., will demonstrate the method by which it was found that PHILIP MORRIS cigarettes, in which diethylene glycol is used as the hygroscopic agent, are less irritating than other cigarettes. Their representative will be happy to discuss researches and problems on the physiological effects of smoking.

Booth 72

ZIMMER MANUFACTURING COMPANY

ZIMMER MFG. CO. will have a complete line of Fracture Equipment on display in Booth No. 72. Among the items of special interest to the industrial surgeons will be the Smith-Peterson Outfit, Reduction Apparatus, Kirschner Outfit, Pelvic Splint, Curry Walking Splint, Forrested Knee Hinges, Phelps-Gocht Osteoclast and the Conzett Goniometer.

Booth 59

WILLSON PRODUCTS, INC.

BRIEFLY, the WILLSON exhibit includes devices which may be classified in two distinct groups: First, apparatus such as the Impinger, Dust Counter, Chlorinated Hydrocarbon apparatus, and heated-therometer anemometer used in the determination or evaluation of the degree of the employees' exposure to atmospheric contaminations. Second, respirators of various kinds worn by workers to decrease or eliminate objectionable contaminants in the inspired air.

Booth 63

PICKER X-RAY CORPORATION

THE PICKER X-RAY CORPORATION will show their Self-Contained Shockproof Diagnostic Unit, consisting of 200-milliampere Four-Valve Generator with Tilting Table and Spot Film Device.

Shockproof Mobile Units of 15 milliampere and 30 milliampere capacity, as well as a new compact Shockproof Portable Unit will also be shown.

Booth 46

ULMER PHARMACAL COMPANY

INDUSTRIAL surgeons are invited to visit the booth of ULMER PHARMACAL COMPANY for a discussion of Proliferol and Phyco Clinical Trusses as used in the injection treatment of hernias. Time and monetary and efficiency losses are eliminated in this method, as the worker is ambulatory. Leading railway and industrial surgeons endorse the method in selected cases, and Proliferol as the desirable solution. Biliposol, the lipoid soluble basic bismuth for syphilis endorsed by Dr. Levaditi of the Pasteur Institute will be featured together with Cal-zo for varicose ulcers, Picro-Benzyl for burns, and specially priced, unusually fine imported haemostats and scissors.

Booth 70

DENVER CHEMICAL MANUFACTURING COMPANY

IN SPACE 70 ANTIPHLOGISTINE will be exhibited. This product, now in its forty-fifth year, is employed by physicians in all parts of the world. There is only one way in which an ethical preparation can attain this distinction, and that is through merit. ANTIPHLOGISTINE is kept on hand and used continually in the emergency hospitals and first-aid stations of many of the great industrial plants, railroads, mines, etc. of this and other countries.

Booth 40

C. B. FLEET COMPANY, INC.

PHOSPHO-SODA (FLEET): An ethical, concentrated preparation of the sodium phosphates buffered and in a stable solution. Phospho-Soda is a reliable intestinal eliminant and detoxifying agent, acting quickly without griping or causing tenesmus. Phospho-Soda (FLEET) has, for 50 years, been recognized by the medical profession as being the desirable choleretic and cholekinetic. The ease of administration and flexibility of dosage gives Phospho-Soda (FLEET) a wide range of usage.

Attractive measuring glasses and samples of Phospho-Soda will be gladly supplied at this booth.

Booth 12

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LOCATED in booth number 12, the MINE SAFETY APPLIANCE COMPANY, of Pittsburgh, Pennsylvania, will display a complete line of safety equipment for the protection of workers in all industries.

A feature of the display will be the M.S.A. Salt Tablet Dispensers, including the "350" and "1500" size and also the new "One-at-a-time" Pocket Dispenser with 24 tablets. The larger dispensers are molded of bakelite, require only one hand to operate, and are weatherproof.

The new Dustfoe Respirator (U.S. Bureau of Mines approved), another M.S.A. product, provides full protection under the most severe dust conditions, and yet weighs less than 3¼ ounces. It will be displayed in addition to the popular Comfo Dust and Paint Respirators.

Other M.S.A. products include Gas Detecting, Analyzing, and Recording Instruments; Goggles; All-Weather First Aid Kits; Skullgard Protective Hats and Caps; M.S.A. Abrasive Masks and Air Line Respirators; and Resuscitation Apparatus, the H. H. Inhalator.

Booths 68 and 69

WESTINGHOUSE X-RAY COMPANY, INC.

THE WESTINGHOUSE X-RAY COMPANY will display new shockproof equipment for high speed chest radiography. A representative exhibit of chest films will also be shown. Probably of greatest interest to the Industrial Surgeon will be a new device for the positive identification of a chest film which will have great value in any legal case.

BOOTH LOCATIONS

4. C. O. SAPPINGTON, M.D., DR.P.H.
6-7. U. S. PUBLIC HEALTH SERVICE.
8-9. HART E. FISHER, M.D, F.A.C.S.
12. Mine Safety Appliances Co.
13. Brooks Appliance Co.
14. Farnsworth Laboratories.
15. LIBERTY MUTUAL.
16. CHICAGO BOARD OF HEALTH.
17. HILGER PERRY JENKINS, M.D.
18. M. H. KRONENBERG, M.D.
26. McManis Table Co.
27. OHIO DEPARTMENT OF HEALTH.
31. Sutliffe & Case Co., Inc.
32. S. M. A. Corporation.
34. DETROIT DEPARTMENT OF HEALTH.
36. NATIONAL SAFETY COUNCIL.
40. C. B. Fleet Co., Inc.
41. MISCELLANEOUS.
42. DoMore Chair Co.
43. JAMES E. M. THOMSON, M.D.
44. Keystone View Co.
45. CARL A. JOHNSON, M.D.
46. Ulmer Pharmacal Co.
49. Fairway Laboratories, Inc.
50. Cameron Surgical Specialty Co.
51. Morton Salt Co.
53. Holland-Rantos Co.
54. NORTHWESTERN UNIVERSITY.
57. JOHN J. PRENDERGAST, M.D.
58. J. B. Lippincott Co.
59. Willson Products, Inc.
60. HARRY E. MOCK, M.D., F.A.C.S.
62. Horlick's Malted Milk Corp.
63. Picker X-Ray Corporation.
64. CLEVELAND J. WHITE, M.D.
66. The Dionol Co.
67. CLARENCE D. SELBY, M.D., F.A.C.S.
68-69. Westinghouse X-Ray Co., Inc.
70. Denver Chemical Co.
71. DeQuinn Physicians Products Co.
72. Zimmer Manufacturing Co.
73-74. JAMES A. JACKSON, M.D.
75. LaMotte Chemical Co.
77. JOHN DEPAUL CURRENCE, M.D.
78. DePuy Manufacturing Co.
79. Philip Morris & Co., Ltd., Inc.
80. AMERICAN COLLEGE OF SURGEONS.
81-82. Eichenlaubs.

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VOLUME 7 JUNE, 1938 NUMBER 6

Silicosis

AS WE have previously remarked, there are many people who now look upon silicosis as an unimportant disease because of its relative infrequency. Preventive measures are proving effective, so that—while it never was as bad as the Hon. Secretary of Labor said it was several years ago (see page 304)—it is, as DR. SAPPINGTON puts it: "Not rare, not common."

DR. REED, on page 314, quotes the National Silicosis Conference figures for 1937: "110,000 have silicosis of some degree. Out of this number 5,000 suffer some form of disablement . . . . 105,000 have silicosis but no disablement." This is not an alarming proportion of 49,000,000 workers—a little more than 0.2%. But it is 10% of the million "exposed to some form of silica hazard;" 20% of the half-million "exposed to a serious hazard."

Thus it is not unimportant, nor a matter of the past. This is particularly true with reference to its cost; and if Wisconsin's experience is any criterion, it is startlingly true. In that state, in 1936, although "noxious dusts," with 71 cases, aggregated only 6.6% of the total number of cases of occupational disease disability indemnified during 1936, the payment of compensation for these cases aggregated $119,737, or 55.4% of the total compensation paid for the entire group of occupational diseases. (See DR. SAPPINGTON'S Table X, page 334.)

Furthermore, silicosis when compared with the chief causes of injury stood second highest in average compensation costs among the fatal cases, first among the relative permanent partial disability cases. It appears, therefore, upon this evidence that the severity of silicosis is the highest of any occupational disease, of which we have adequate record at the present time.

And the severity is directly reflected in the cost. Table XII, on page 334, is illuminative: 13 cases of lead poisoning at an average indemnity paid per case of $105; 150 of dermatitis at an average of $51; 22 of silicosis at an average of $5,225.

This is going from newspaper to check-book with a vengeance!

The Formula!

SPEAKING of newspapers, their press services, and even some of the news magazines, *Time* and others, have been importuning DR. J. R. GARNER to disclose the formula for his APSA the apparent effects of which were described, and illustrated, in our May issue under the heading: "Cancer — A New Treatment: Holding Much Promise."

To all of these he has replied about as he wrote to us:

"I presented two cases before the Fulton County Medical Society Thursday evening, May 19, 1938, and quite naturally this was the subject of much discussion among the doctors in Atlanta yesterday, and I find, as is to be expected, that the comments are both favorable and unfavorable. Many seemed to take the attitude that I should at once make known my formula. In the first place I have worked on this formula for seven years before bringing it to the present state. I do not feel that it is absolutely perfected at the moment. The process of manufacturing APSA is very intricate. It cannot be manufactured by any prescription clerk in a drug store, and I fear that if I made known the formula now, some physician would attempt to mix up some kind of concoction containing the same ingredients and meet with disastrous results — which would throw opprobrium upon my entire work. Hence I feel that until I can assure everyone that it is positively a cure, and until I can perfect the formula and it can be made more stable than it is at present, I must decline to let it become generally known."

This seems entirely reasonable. DR. GARNER said in his May article: "Additional experimentation is being conducted with a view to overcoming this difficulty (the volatile property of one of the ingredients and the resultant instability of the compound) and very shortly making it possible to publish the formula and entrust its manufacture and distribution to some reliable pharmaceutical house."

Occupational Diseases and Industrial Surgery

Comparative Values of Injection and Surgical Treatment of Hernia

GEORGE EARL, M.D.,
Saint Paul, Minnesota

PREVIOUS to the last decade, injections for the cure of hernia attracted little attention.* The solutions of Pina Maestrae, of Spain, were handled with great secrecy and in this country sold at such high prices that they had been little used. Mayer of Detroit was using the injection method long before he gave medical publicity to it. Bratrud, of Minneapolis, together with McKiney, Rice, and Larsen, deserve credit for taking the matter up in a clinical teaching way and giving the profession solutions, methods, and the results of their experience.

It is estimated that 10% of the population have hernia in some form or other. The majority, influenced by economic considerations, necessity of hospitalization, objections to surgery and recurrences after operation, prefer to wear trusses. The tendency now is for active treatment to be sought earlier.

Inguinal hernia comprises most of the cases selected for injection because trusses are fitted more easily, and, anatomically, the injections are simpler. The direct inguinal type gives more difficulty than the indirect because of gravity and also because the structures of the internal and external oblique muscles are often frayed out or almost absent. Evidences of this are the many surgical devices used, such as fascia lata strips from the thigh, turning down of fascia from the rectus muscle or incision of the fascia of the internal oblique near its attachment to the rectus muscle so as to free the internal oblique for approximation of Poupart's ligament. Direct hernia becames more frequent in later life. Age presents poorer reacting qualities, hernia of longer duration, degenerative changes, often increased fat, and the protruding abdomen placing the inguinal hernia more at the bottom of a sack. The number of injections needed increases with each decade of life, and many more are usually needed with direct than with indirect inguinal hernia. The same factors necessitate more careful surgical procedure with direct hernia.

IS INJECTION likely to damage the spermatic cord or the circulation of the testicle? Before any treatment, surgical or injection, the status of the cord and testicles should be carefully noted. Variation in size may be developmental or due to illness, such as mumps, or it may follow operations. I am injecting a dentist who suffered marked testicular atrophy following an operation and, with hernial occurrence on the other side, he has selected the injection method. The spermatic cord is very resistant to any needle inplantation. The younger the individual, the more resistant the elements of the cord are. The thickness of the wall of the cord on section is many times greater than the lumen. Records of potency, semen examinations, findings at such operations as may be necessary following failure of the injection method, and post-mortem examinations in operated and injected individuals will ultimately determine comparative risks.

Femoral hernia, if reducible, may lend itself to injections. Femoral hernia occurs jointly with inguinal hernia more frequently than is usually suspected Sometimes it is not noticeable until the inguinal hernia has been corrected by operation or injection.

Umbilical hernia, if small and free from any abdominal contents, may be treated by injections.

Post-incisional abdominal hernia, if small, may occasionally be closed by injections. One difficulty is that often this type of hernia may have multiple points of weakness. In the upper abdomen, because of the presence of the mesentery and liver, there is less chance of the development of bowel adhesions and hence injections are safer above the navel than below. Unless very minute and treated early, post-incisional rectus muscle and midline herniae below the navel are positively surgical.

Recurrences in inguinal hernia following operation often lend themselves to control from injections. Previous operation has rearranged the tissues so that injections may fill in weak spots more readily.

Before injections are made, a satisfactory truss must be fitted. No one type fits every kind of hernia. A spring bar is most efficient in the inguinal type. In umbilical and incisional hernia, trusses with attachments encircling the body are used. The truss may have to be adjusted repeatedly. The surgeon should fit and closely observe the truss. The truss must function positively to hold the hernia. For the discomfort and chafing present at times, a buffer cloth between the truss and skin is helpful. Toughening of the skin with alcohol and dusting powder is useful. The truss is worn night and day to begin with and preferably so during the injection period. After apparent cure, it should still be worn two to six months longer, depending upon the type of hernia and the difficulty in securing healing. Then it is left off at night only and later during the day unless special strain or activities are undertaken.

Next comes the choice of the irritant to produce a foreign body granuloma—microscopically, a proliferation of endothelial and connective tissue cells forming scar tissue. Many solutions are being used, and this fact in itself is evidence of the still unsettled question as to which solution is best. Of the many in use, I have had experience with five.

1. THUJA MIXTURE

Phenol, 50 parts.
Alcohol, 25 parts.
Lloyds Specific Tincture of Thuja, 25 parts.
Allow to stand two days and then either decant or filter.

Except for a half dose at the internal ring, an average of eight minims is injected at each treatment. A tuberculin syringe is used. With such a small quantity of solution only a small area is affected, necessitating a greater number of injections. The Thuja mixture is its own sterilizing agent and needs no preliminary novocain for anesthesia.

2. MAYER FORMULA

Zinc Sulphate, 1 dram.
Glycerin, 4 fluid drams.
Fluid Extract Pinus Canadensis (dark), 5 fluid drams.
Phenol Crystals, 6 drams.
Aqua Cinnamomi, 1 fluid dram.
Sterlized chemically pure redistilled water, 2 fluid ounces.
The solution must be brought to boil each time before it is used. The average dose is 8 minims.

3. BRATRUD FORMULA

Tannic Acid 0.50.
Benzyl Alcohol 3.00.
Thymol 0.50.
Alcohol (95%) 100.0.

This product is suggested by Dr. A. Bratrud, of Minneapolis. The tannic acid may be increased to 1% in selected cases. Its advantage is that a larger amount can be used, probably on the average of 2 cc. It is preceded by 1 cc. of 2% novocain. Bratrud adds two minims of Thuja mixture. All solutions are given through the same needle with change of syringes. The larger amount of solution gives a reaction over a larger area.

4. SODIUM MORRHUATE

This solution was suggested by Dr. D. D. Turnacliff, of Minneapolis. I have used 2 cc. of the solution preceded by 2 cc. of 2% novocain and followed by 1 cc. of novocain. I have limited its use to the above dosage in difficult cases in which other solutions

(Continued on page 355)

* In *Minnesota Medicine*, April, 1938.

THE AMERICAN ASSOCIATION

of

INDUSTRIAL PHYSICIANS and SURGEONS

Component Societies

Central States Society of Industrial Medicine & Surgery

Association for the Advancement of Industrial Medicine & Surgery

THE object of this Association shall be to foster the study and discussion of the problems peculiar to the practice of industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes and results of the medical care of employees, and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for the mutual advancement in the practice of their profession.

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ANNUAL MEETING

THE 1938 Annual Meeting of the American Association of Industrial Physicians and Surgeons, and the 1938 Midwest Conference on Occupational Diseases, will be held at the Palmer House, Chicago, June 6, 7, 8 and 9, 1938.

Proposed Amendments

IT IS proposed to amend the Constitution and By-Laws of the American Association of Industrial Physicians and Surgeons as follows:

Article III. Sec. 1: Membership:

Membership in this Association shall be of four classes:

(a) Active: Only physicians who are actively engaged in the practice of Industrial Medicine and Surgery or who are engaged in the investigation of industrial medical problems shall be eligible to active membership.

(b) Associate: Other physicians and individuals who are not physicians but whose work or activities bring them within the realm or province of the industrial physician, shall be eligible to associate membership.

(c) Honorary: Any person who has contributed distinguished service to the objects for which the Association stands shall be eligible to honorary membership.

(d) Fellowship: Active members of the Association and of its component societies, after two years of membership in the Association or their respective societies, may apply for Fellowship. Fellowship will be conferred by the Board of Directors upon applicants who have been actively engaged in the practice of Industrial Medicine for at least five years and shall successfully pass an examination to determine that they are fully qualified to practice Industrial Medicine and Surgery.

A suitable certificate, attesting that they are "qualified in the Art and Science of Industrial Medicine," will be issued by the officers to those upon whom Fellowship has been conferred.

Amend the By-Laws as follows:

Article II. Sec. 1: Dues:

In line two, after 'per year', add: "except where a special arrangement is made with an organized group to come in as a body."

Article V. Committees: Add Sec. 4:

The President shall appoint an Examining Committee to pass upon the qualifications of applicants for Fellowship.

A further amendment is offered, to change the beginning of the fiscal year of the Association from January 1 to July 1, of each year.

These proposals will be presented, for appropriate action, at the Twenty-Third Annual Meeting, in June, 1938.

—*Continued from page 353.*

had previously been used. In my experience, with the dose mentioned, it gave the most marked reaction of all and therefore should be used with caution. I have seen no other reports regarding its use.

5. SODIUM PSYLLIATE

Of late I have used a sodium psylliate preparation. It causes no damage if spilled on the skin, the reactions are certainly less marked and, experimentally on animals, the reactions even on the peritoneum are reported mild. In cases which are difficult to close, I am inclined to return more frequently to solutions of greater reaction, such as Bratrud's formula, sodium morrhuate, or Thuja mixture.

For infection of an inguinal hernia, the patient is placed on a table with the hips elevated. Alcohol (70%) is applied to the skin area. The approach to the region of weakness is through skin, fat, and fascia. The resistance of the fascia is definite. When the needle is passed through the fascia, freedom of motion of the needle is evident. An attempt should always be made to withdraw the piston of the syringe, to insure avoidance of vessels. The patient's reaction to pain also guides in the placement of the needle. The injection should be immediately stopped with any unusual reaction. It should be given slowly. Repeated injections are placed in various areas of the inguinal canal according to the reaction obtained and points of greatest weakness. The nearer the internal ring, the more need for caution.

The frequency of injection depends upon the reaction, upon the number of areas needing injection and, to some degree, upon the convenience of the patient, particularly an out-of-town patient. The injections range from daily to weekly injections, usually allowing a week to elapse between injection of each area. Reaction in the form of swelling is encountered. I have seen none that has not subsided. I have not encountered infection.

In umbilical hernia, the defect is approached from all sides, dosage at any point being from one-quarter to one-eighth of that used in inguinal hernia. The peritoneum should not be penetrated. In femoral hernia, the dosage is about one-quarter and the approach is made with great care. Incisional herniae, except in the inguinal region, are approached with much caution.

THERE are many interesting reports in literature. Pina Maestrae, of Spain, reports favorably on 8,000 cases.

Mayer, of Detroit, states that in his work a census of 2,100 cases of inguinal, femoral and umbilical hernia by subcutaneous injection showed complete relief without recurrence after a period of years in 98% of the cases. Furthermore, more than 200 case of hernia recurring after operation were subsequently completely

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1937 - 1938

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relieved by the subcutaneous injection.

Bratrud and McKinney, at the University of Minnesota Hospital, and Rice and Larson, at the Minneapolis General Hospital, have given favorable reports on large series. This work at the University of Minnesota is especially to be commended because of the spirit of open-mindedness shown, and also because of the scientific approach and willingness to teach. Samuel W. Fowler, of New York City, has recently reported several hundred cases. Total reports now from all sources are large.

During the years 1933 and 1934 in a private practice trial, I operated upon 38 patients and injected 42. Since then over two years have elapsed. Patients treated since 1934 are too recent for evaluation. Of the injection cases, 37 were inguinal hernia, two were incisional, one umbilical, and two femoral. Of the 37 inguinal, four had, in addition, femoral ring impulse; hence, both areas were injected. Two of the inguinal group had marked hydroceles which were treated also. Three of the injected cases were surgical recurrences, one of which had had three operations, and another two operations. These operated cases reacted well, as the operations had left smaller areas of weakness for closure.

Of the operated patients, two had undescended testicles, two had marked testicle atrophy from mumps (one unilateral and one bilateral), five had femoral hernia and six had post-incisional hernia. The remaining 23 of the surgical group were inguinal herniae.

CERTAIN difficulties were met:

1. Severe enough reactions to cause the patient to lay off from work. The first was a laborer doing exceptionally heavy lifting and to whom I gave the largest dose of sodium morrhuate I ever used at one injection. He had the most marked reaction of my series. His lay-off lasted one week. He had a closure of the hernia following the severe reaction and has since referred a patient to me. The second was an older man who complained much of the character of his work. Besides compensation, he had other sources of insurance income and had it not been for the character of his work and also for his insurance, I feel that he would not have laid off at various times for a total of several weeks. Three other patients laid off, missing part or all of a day from work. Since using sodium psylliate, none has laid off from work. Reactions have been much reduced, but I am of the opinion that more injections are required than with the other solutions.

2. The most difficult herniae to close were in four patients, all old men with long standing, large, direct herniae. Two I have operated upon after prolonged injections. Adhesions were marked from the injections, but operations was not more difficult than expected. One of the two tired of injections and suggested operation him-

self. Another I apparently closed with 44 injections, but the hernia reopened. I have advised operation, which he may accept, although he still comes for injections. Another has had 39 injections and progress is very slow. He comes occasionally for injections. The advice I would give generally for hernia of this type is plastic repair. Others of this type, which apparently closed, will have to be injected again from time to time for recurrences or they may come to operation by me or drift into other hands, if they have not already done so. Observance for recurrence should be repeated regularly. Extra injections after apparent closure are necessary to improve results.

3. Swelling in the tissues accompanying the cord does occur at times with all the solutions mentioned except sodium psylliate. It has always subsided, however, in my experience. Twice I removed fluid from the center of the reacting mass. Patients become somewhat disturbed over such swellings and must be reassured.

4. Pain results from proximity to the nerves or to the peritoneum. In such cases the needle must be reinserted.

5. Influx of blood with a preliminary withdrawal of the piston, previous to injection, has occurred several times. The needle was promptly withdrawn and no complications followed. If the withdrawal had not been made promptly, trouble might have followed.

6. With patients who are unusually temperamental and highly sensitive to any manipulations, bromides or phenobarbital reduce sensitiveness.

7. Difficulties with operation due to adhesions following injections. I have operated on two patients whom I myself have injected. Further, I have operated upon two others who had been treated elsewhere. Recently I removed an acute appendix in a patient who had had injections previously by a very competent man. I found the cecum markedly adherent, which made the operation for an acute, beginning gangrenous appendix located postcecally most difficult. Of course, at times, we find marked adhesions of the cecum without the patient ever having had injections, but in this case the adhesions were directly in line where the injections had been placed. Unquestionably, patients whom I have injected will have some adhesions which will come to light at operation or postmortem.

The cases reported on were injections begun in 1933 and 1934, and during those years, taking in all types of herniae, I injected a few more than I operated upon, the ratio having been surgical 47.5%, and injection 52.5%. Of the cases of hernia I have seen in 1935, 1936, and 1937 and have not reported because they are too recent to evaluate, I have operated upon more than I have injected, the ratio being surgical 57% and injection 43%—a distinct shift toward surgery. Just where the pen-

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dulum between surgery and injections will come to rest will probably depend upon the individual surgeon. Some surgeons will operate only, others will tend toward injection. The majority of surgeons will probably do both injection and operation, depending upon the indications, upon the surgeon's inclination and experience, and somewhat upon the patient's economic circumstances and acceptance of, or objection to, surgery. A factor to be considered is that in the future patients with herniae will come earlier for treatment due to compensation insurance, better health education, and the interest aroused by the injection method.

Conclusions

HERNIAE to be injected must be reducible and satisfactorily held in position with a truss. The large percentage of injections will be made in inguinal hernia, a few in femoral and umbilical. Only the smallest postoperative abdominal herniae above the umbilicus lend themselves to the injection treatment. Below the navel the small bowel is more commonly directly adjacent to the peritoneum. If surgical recurrences are found early enough, the injection method may prevent reoperation, particularly in inguinal hernia.

Of the solutions used in my experience, sodium psylliate has been less accompanied by pain and swelling. Proliferol and sodium morrhuate produce greater reaction but, in spite or that, may be indicated. Thuja mixture and Mayer formula are useful at times. Other solutions with which I have had no experience may have equal or greater merit. Study of injection material will be continued and widened.

Because many injections are being given to older men who are succumbing to the degenerative diseases, postmortems should be increasingly available. Study should be made of the question of semen and potency in patients operated and in those injected, and the grouping of complications from either operation or injection increasingly recorded.

More accurate observation of the condition of the testicle and cord, previous both to operation and injection, should be recorded. It is natural that great interest should be aroused with any change in these organs, but too often an unfair and unjustifiable attitude of complaint is taken and hence every precaution is needed to record previous abnormalities.

Precautions always to be remembered are: Complete replacement of protruding tissues, which should positively be held in place with truss; withdrawal of the piston of the syringe to avoid injecting into a blood vessel; slow injection to try out the location and patient's reaction; careful follow-up and extra injections to secure sufficient strength in the holding tissues.

In selecting cases and advising pa-

tients concerning results with either operation or injection, ease of cure is is in proportion to the decade of life, size and character of the hernia, general condition of patient's health, patient's cooperation, and the doctor's interest, knowledge, and experience in the procedure. A simple, indirect inguinal hernia is relatively easy of closure by the chemical injection method. All other types of hernia deserve careful study before treatment, other than operation, is advised. In the aged or decrepit, injections may bring relief without cure.

A qualified enthusiasm for the injection method will, of course, secure better results than half-hearted interest and less experience. To secure results with the injection method takes more persistence, close attention to detail and study of the individual case than some are willing to give when operation offers satisfactory results.

Because of recurrences and realizing that, after all, only scar tissue is secured from injections and this tends to give way, and because of the other difficulties discussed, I tend increasingly to advise operation more often than when first I began the trial of the injection method.

Touch Typewriting with One Hand

DURING my experience as teacher of typewriting I have had students of many types, of differing physical and mental makeup, of varying degrees of ambitions, indifference, courage, and decision. I have had students who relaxed naturally both physically and mentally, students who were insistently tense in mind and body; students minus a finger, a thumb, or with some minor deformity of the hand; students with long slender fingers, short stubby fingers, nimble fingers, stiff awkward fingers, light hands, heavy hands, tall students, short students; students with long arms, and students with short arms. Remedies for whatever difficulties were encountered incident to these varying conditions were available or quite readily found, and the difficulties disappeared.

But it had not fallen to my lot to devise ways, means, and methods for teaching touch typewriting to a one-handed student until I was asked to take Mary Ranney into my class. Mary has a good right hand, but the left is wholly lacking. My first thought was that it would be quite impossible, but the proposition interested me and upon second thought I felt that it could be done and was willing to try. It meant that an entirely new method would have to be devised with no foundation, precedent or experience upon which to work. So far as I know it had never been tried, and inquiry of all sources from which I thought help might be obtained brought no helpful answers and little encouragement. After some hours of thought I arrived at tentative plans which, if perfected, I believed would work successfully. I took Mary into my confidence, the plans were finally completed, and we were ready to proceed. Mary's naturally cheerful attitude toward the undertaking, and her willing response and cooperation gave me courage and assured success. Her experience and resourcefulness in doing things with her one hand that regularly required two hands were, of course, important.

Further to Mary's credit she has all the time carried the work of another course. Her typewriting has been an addition to a reasonably full schedule, and some of the time it has been difficult for her to find time for her daily lessons and practice. She has worked under school difficulties not experienced by regular typing students.

At the time this is written after 21 weeks' instruction two hours daily, she has attained a gross speed of 42.8 words a minute for 15 minutes with but four errors, on official test matter furnished monthly from New York by the typewriter company. After penalizing her 40 words for the four errors according to international contest rules, she has a net speed of 40.1 words a minute, receiving the official award granted by the typewriter company. It is interesting to note that this is more than one-fourth the speed per minute of the world's professional champion typist. She has used the same text, practiced the same lessons, and completed the same assignments according to the regular class schedule, and meeting the same requirements as other students. The only difference has been in the operating technique, she following her individual charts and using one hand to manipulate the entire keyboard, while the other students have followed the charts in the test and using two hands.

Mary has now completed her commercial and banking course and is beginning the stenographic and secretarial course. At the time she entered the Cedar Rapids Business College it would have been considered altogether impracticable for her to undertake this course, because of the seeming impossibility for her to at-

* Ivy L. Rady, Cedar Rapids Business College, Cedar Rapids, Iowa, in *Rehabilitation Review*, Vol. XI, 1937, Nos. 11-12.

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tain any measure of proficiency in the operation of the typewriter. I look forward to materially greater proficiency in touch typewriting as she progresses in this course, and I predict a surprising record in speed, accuracy, and all-around efficiency by the time the course is completed. In any event the success of this undertaking has proved that speedy and accurate touch typewriting with one hand is possible, and not necessarily difficult. At least it has not been difficult for Mary, she has enjoyed the experience, and it has been a source of much satisfaction to me.

With the thought that my experience in this case may be of some help to some one else, I am, at the request of Mr. Grant, very glad to give an outline in brief of my method of procedure. This was necessarily original on my part and was made to fit this particular case. It has proved successful. It may at least serve as a suggestion for similar cases and possibly for cases differing from this one. It would probably have to be modified more or less to meet difficulties peculiar to the individual.

The Technic

THE fingers of the right hand are placed on the center of the keyboard over fghj for home positions. The oblique sections of keys are associated with each finger as follows: the first finger strikes vfr4; second nger, bgt5; third nger, nhy6; and fourth finger, mju7.

When striking keys with first or second fingers, the fourth finger hovers over j as guard for home positions. When striking with third and fourth fingers, the first finger hovers over f as guard key. These guard positions are very essential, especially when the first and fourth fingers stretch over to three and four rows beyond the home keys.

All three oblique divisions of keys on the left side of f are struck with the first finger. The reach for each key is made from home position f. The remaining four oblique sections of keys to the right of j are reached by the fourth finger. The location of each of these keys is found from home position j.

Though all of these keys struck with either the first or fourth fingers are reached from home position f or j, the mind thinks of these keys in the natural sections of the keyboard. For instance, when the reach for w is made, the first finger moves to the row above and two sections to the left of f, and the mind still associates w in the oblique section of xsw2. The relative position of one key to the other is quickly remembered when the keyboard is learned in these natural divisions.

This student has her right hand and this method is adapted to the use of that hand. A person having only a left hand would use a similar method excepting that jhgf would be the home positions for fingers 1, 2, 3, and 4 respectively. All keys to the right of j would be struck with the first finger, and all keys to the left of f with the fourth finger.

The paper release lever is pressed down just before the hand places paper back of platen. When the lever is released, the paper is caught far enough under platen so the hand is free to grasp cylinder knob and twirl paper farther into the machine.

Miss Ranney's left wrist is used on the left carriage return lever of the typewriter. This wrist also operates the back spacer, key lock release, and the left shift key for all capitals and other characters. The right shift key is not used. The difficulty of depressing the shift key with the wrist without interfering with surrounding keys has been eliminated by building up the key to the height of about an inch. A pedal could be used by a person with one arm.

"Social Uplift is Stooge Act"

ACCORDING to one of the endowed pinko weeklies Dr. Morris Fishbein is one of the principal sachems of a medical Tammany Hall, the American Medical Association.* Doc. Fishbein in the regalia of a Tammany sachem would add something to the local color of Chicago's near North Side, but it took Greenwich Villagers to think that one up. The headquarters of the association has little about it to suggest a political headquarters.

No one ever got a drink there even under the 18th, when doctors were a privileged class, assured of a minimum income by their Volstead law permits to write prescriptions.

I guess that Fishbein can take care of himself in any battle with the pinkos and I know nothing of the merits of the controversy between orthodox medical ethics and "social medicine." But I can't help noting certain implications of a paragraph in the pinko punko publication.

It says: "A special committee of the New York State Medical Society has formulated a program calling upon the government to provide medical care and health insurance for the indigent and lower income groups." There we have the same old line—"program," "government aid" and "groups." Take those cliches out of uplift patter and New Deal propaganda and what would be left?

BACK of the words and phrases lie certain assumptions that spell evil not good for the poor. While folks are poor, no red-blooded human being can object to helping them, and I can't get excited about whether the necessary help is to be paid for by government or private citizens.

What annoys me is the patronizing unctiousness and sanctimony that pervades all this alleged philanthropy and uplift, I don't care whether it is part of a fireside chat, a paragraph in the report of a tax-dodging foundation left by some financial buccaneer, or one of Mme. Perkins' communiques.

Having been born in a county where there was nobody worth more than $20,000 and no propertyless people, except a few half-wits at the county farm, I suppose I am a hopeless anachronism, a hick who has never yet brushed all the cockleburs off his pants. But neither have I ever got around to the idea that there is any reason for people to remain poor in America or that it is necessary to have static social "groups."

WHEN I hear people going on about their great love of the poor, I can't help recalling old Victor Hugo's crack that the only good the rich could do the poor would be to get off their backs. And today I think that goes quite as aptly for politicians, philanthropists, social workers and the whole kit and kaboodle of the "social minded."

If this country would go back to real Americanism, the most radical social creed on earth as exemplified by Jefferson, Jackson and their kind, we would have no need for "social medicine," " social security," or any other of the "social" abominations of the New Slavery that Herbert Spencer warned us against 50 years ago.

The people of the great cities of America today, of all the races and creeds, are just as capable of self-help and self-support as were the men and women who started this country. I say that after having looked 'em over in plenty of cities. Nor do they want to be nursed, coddled and patronized.

Back of the social workers, the foundationeers, the professional philanthropists and all who have developed into beneficiaries of a vested interest in poverty and dependence are the grab-it-alls whose vested interests and special privileges depend upon the limitation of production and the curtailment of opportunity and competition. Today the cost of relief and the social uplift are the price paid for monopoly. The uplift is a stooge act.

Open the throttle of the American economic engine and the people of America will have no need for "social medicine." They can do like their daddies did—hire the doctor of their choice.

Useful for Burns

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* HARPER LEECH, in *Chicago Daily News*, May 13, 1938.

* B. FANTUS and H. A. DYNIEWICZ in *J.A.M.A.*; quoted in *Digest of Treatment*, May, 1938.

Newer Treatment of Burns

R. O. JOPLIN, M.D.,
Louisville, Kentucky

IT IS estimated that 6,000 persons lose their lives every year, and many thousands more are disfigured or temporarily disabled as the result of burns.* The local treatment of burns has never been standardized. Today the methods are many and varied. This was recently brought out in a survey of records of a large insurance company. Twelve hundred cases of burns collected from 31 states were treated by 386 different surgeons, who used 47 different types of treatment. I question whether any other type of injury or disease has such a variety of therapeutic agents. The majority of these cases were probably ambulatory, which accounts for the variety of treatment.

Injuries due to burns can be divided in two classes: (a) less extensive injuries in which the problem of toxemia does not arise, (b) severe and extensive injuries where life is endangered by shock and the onset of acute toxemia. Fortunately the first class is the one most commonly seen, and can be treated at the office or home. In caring for these cases one should employ methods that meet the following criteria:

(1) Simplicity of application and infrequent changing of dressings;
(2) Alleviation of pain;
(3) Control or eradication of sepsis;
(4) Minimal overgrowth of granulation tissues;
(5) Absence of local skin irritation, dermatitis or general toxic symptoms;
(6) Rapid and sound healing without contractures.

The question arises as to what to apply to the burned area. Ointments of various composition are still the favorite therapeutic agent, probably because some type of grease has been applied before the patient is seen by the doctor, and also because ointments are most always present. Five per cent tannic acid, tannic acid jelly, 5% aqueous solution gentian violet, tannic acid gentian violet mixture are very good, but require more time for application and thorough removing of all grease before being used. Stewart, of Edinburgh, advocates the use of amyl salicylate plus a phenol antiseptic for minor burns in the form of a wet dressing after first cleansing the wound with saline, then covering with wool and a dry dressing; the dressing should be changed every fourth day, and swabs soaked in the solution again applied, the dressing next to the wound not being removed until healing has taken place. The application of tannic acid jelly to get the benefit of coagulation followed by subsequent dressings saturated with camphorated oil has given gratifying results; reepithelization is especially rapid as there is no secondary infection and no trauma associated with the change of dressings.

The treatment of the second class of cases is more difficult, but the therapeutic agents have been narrowed down to a chosen few. Whether one believes in the toxic or plasma-like fluid loss theory as the cause of a secondary shock in burns, two facts stand out:

1. That secondary shock is one of the chief causes of death, and
2. Tannic acid seems to be effective in combating shock when present, therefore it matters little whether it acts by precipitating toxins or by sealing off the capillaries, and preventing fluid loss.

The important thing is that shock is combated and the death rate is lowered. In burns as in all other traumatic conditions our first concern should be the treatment of the patient, then the treatment of the local condition. By treatment of the patient we mean:

1. Sufficient morphine to keep him comfortable.
2. Remove all clothing, put patient to bed and keep warm, preferably with a cradle light.
4. Force fluids by all available routes to combat shock.
4. Later blood transfusions may be given to combat infection, toxemia and anemia.
5. Tetanus antitoxin should always be given when indicated.

Treatment of the local condition can be divided:

1. Into various dressings, antiseptics and solutions, as ointments, vaseline, paraffin, carron oil and cod liver oil dressings, soda bicarbonate, saline, picric acid, horse serum and Dakin's solution.

2. Methods depending on protein precipitation, as tannic acid, tannic acid jelly, tannic acid plus antiseptic base, gentian violet, brilliant green and ferric chloride 5%. We are familiar with the tannic acid and gentian violet treatment, and papers on these treatments have been given.

Brilliant green, another anilin dye is said to be more efficacious than gentian violet, and less destructive to the germinal epithelium than tannic acid. In using this dye the surrounding skin is painted with a 1% solution of brilliant green in 60% alcohol, the wound thoroughly cleansed and the burn painted with a 1% aqueous solution. After three to five days the alcohol solution can be used on the wound, the burn is covered with a thin layer of gauze, and applications made once or twice a day. After a few days the solution is substituted by brilliant green jelly which is allowed to dry on the wound for five to 10 minutes, and then covered with a thin layer of vaseline gauze; this gauze is changed every two to four days, depending on the amount of secretion. The tannic acid silver nitrate treatment advocated by Bettman appears superior to other methods we have at present. The advantages listed by him are as follows:

1. The saving of lives that would be lost through the slower method of tanning.
2. The immediate stopping of the loss of body fluids, thereby preventing the consequent concentration of the blood.
3. The immediate prevention or very definite minimizing of shock.
4. The immediate prevention of the absorption of toxic products.
5. The prevention of infection by the short period of application of moisture and the early drying of the tanned tissues.
6. The saving of the kidneys and other organs from the effects of fluid concentration and the absorption of toxins and infection.
7. The greater comfort of the patient.
8. The fact that the patient is carried safely past the first 24 hours, the most critical period following a serious burn.
9. The fact that the patient avoids the second critical period, that of infection and late absorption of toxic products.
10. The simplification of the nursing problem, especially in the first 24 hours.
11. The prevention of further breaking down of tissues, resulting from long application of wet dressings.
12. The prevention of chilling, resulting from the long application of cold, wet dressings.
13. The formation of a thin, flexible coagulum.
14. The speedy healing of the burned areas with a shortened period of hospitalization.
15. The prevention or minimizing of heavy contracting scars by early rapid healing in the absence of infection.
16. The lessening of the amount of skin grafting and secondary corrective surgery.

THIS treatment should be carried out as follows: *first*, the wound should be thoroughly cleansed, and any grease or oil removed with ether or benzine; *second*, all blebs opened and loose skin and burned tissue removed; *third*, a thorough application of 5% tannic acid solution is made with cotton swabs, and following this a 10% silver nitrate solution applied in the same manner—coagulation takes place in a few minutes; *fourth*, place the patient under a light cradle so the burned areas will be kept dry.

*Read before the Jefferson County Medical Society; published in *Kentucky Medical Journal*, April, 1938.

The Importance of Pre-Employment and Periodical Medical Examinations

—Summary of Medical Findings in 500 Cases—

ROBERT C. PAGE, M.D.,
Chicago

THE revision of the occupational disease law in the State of Illinois has done much for the general welfare of all concerned.* The employer, becoming aware of possible occupational disease exposures existing in his plant, is following modern engineering methods, as a means of control. Likewise, he is realizing the importance of having robust and healthy men in his employ. In relation to occupational disease, he does not wish to assume unnecessary liability by employing a man already suffering from a known occupational or disabling disease. In order to prevent such occurrences as well as to obtain the highest degree of work efficiency, all prospective employees should receive pre-employment medical examinations whether they are to be exposed to a hazardous environment or not. The benefits to be derived from such a program I shall exemplify later. One outstanding feature is that it reaches a group of the population who under other circumstances would not receive a medical examination, whether it be from a personal or financial reason.

To briefly exemplify my previous statement relating to the benefits that may be derived from the routine examination of all employees, I have prepared Table 1 on which are summarized the results of 500 complete medical examinations. (See page 363.)

All had the same type of examination which included a previous occupational and medical history, a complete physical examination (x-ray of chest included as well as laboratory work in the nature of urinalysis, hemoglobin determination, and Wassermann and Kahn test for syphilis). As these examinations were made by myself and all of the films read in conjunction with one roentgenologist,[1] the conclusions derived are the results of one system of thought.

In this study the following industries are represented: foundries, both iron and brass, battery and printing plants, tool shops, novelty makers, bicycle works and glass manufacturers; and thus it furnishes a bird's eye picture of the medical status of employees of many of our smaller manufacturing establishments throughout the city.

The study includes:

(a) One hundred men between the ages of 17 and 35 who received pre-employment examination, and who at the time of this examination denied previous occupational disease exposure.

(b) One hundred men between the ages of 35 and 73 who during their whole life according to occupational history had never been subjected to a hazardous exposure.

(c) Three hundred men who during their working life have been exposed to an occupational disease hazard—dust for the most part, with lead in specific instances. This group includes 100 foundrymen, 20 sandblasters, 30 battery plant employees and 150 men who follow such trades as polishing, buffing, grinding, asbestos workers, etc. Nearly all nationalities are represented.

Let us first consider the 100 new employees with an age range from 17 to 35; of these men 96 are American born, three are Germans, and there is one Austrian. Twenty-five per cent of this group are apprentices for polishing, buffing or grinding; 8% are punch press operators, the remaining group includes such occupations as tool and die makers, lathe workers, machinists, welders, assemblers, tool clerks, inspectors, time keepers, etc. No unhealthy chests were detected in this entire group. The physical status was regarded as excellent in 81 cases, average in 17 cases, two men were rejected on account of bilateral inguinal herniae. Other physical defects recorded were: poor teeth nine instances, impaired vision in three cases, hypertension one instance and trace of albumin in the urine in two instances. This group illustrates the type of individual from a physical standpoint that is chosen by an employment man as a prospective employee. Six manufacturing establishments are represented in this group.

Let us now visualize the possible physical status of these men 15 to 25 or even 35 years from now. No protective measures or better working conditions are needed in this instance as these men are not subjected to an occupational disease exposure.

I now refer to the 100 men examined between the ages of 35 and 73 who according to their occupational histories have never been exposed to an occupational disease hazard. Twenty per cent are tool and die makers, 13% are machinists, the remaining group includes clerks, assemblers, maintenance men, punch press operators, engineers, millers, sheet metal workers, inspectors, janitors, blacksmiths, salesmen, laborers and wood workers. Of this group 50% are American born, 20% are Germans, 7% are Polish, the remainder have emigrated from the other European and Asiatic countries.

With each decade the physical fitness is lessened with a corresponding increase in the physical and systemic defects: between the ages of 36 to 45 yars, 43.6% of the men employed are considered to be in excellent physical condition, 54% are classified as average, 2.4% are below average, as compared to 24.2% graded as excellent, 69.9% classified as average and 5.9% as below average in the age group of 46 to 55 years. From 55 to 73 years a study which included 12 employees, 8.3% are classified as excellent and 91.7% as average. The cause of this fall in physical fitness is explained by the increase in physical defects, many of which could be either prevented or treated if medical attention is sought in time. I now refer to eye conditions, particularly in the nature of poor vision, where there is a percentage incidence of 10.9% in the 36 to 45 age group, 24.2% in the 46 to 55 age group, and 8.3% in the 55 to 73 age group. Oral hygiene is generally very poor. Dental attention was needed in 21.9% of cases in age group of 36 to 45, 30.3% in age group of 46 to 55 years, and 41.5% in group aged between 55 and 73 years. Other physical and systemic defects in the nature of high blood pressure, herniae, varicosities, syphilis, diabetes, kidney disturbances, etc., which help to account for the change in physical rating will be discussed in more detail later. As you will note, lung conditions play practically no role in this group.

The group of 300 employees who have been exposed to hazardous occupations for varying periods during their working lifetime, for purposes of discussion will be considered collectively with special refernece given to any unusual feature in any one group whether it be sandblasters, foundrymen, or polishers. In the age group from 17 to 35 years, 58.5% are classified as excellent, 38.7% as average and 2.5% as below average, while between the ages of 36 to 45 years, 17.6% are classified as excellent, 76.4% as average, 4.8% below average, and 1.2% questionable. From the age of 46 to 55 years, 5.2% are graded as excellent, 75% as average, 18.6% below average and 1.2% as unfit for continued employment. Twenty-eight employees make up the 56 to 70 year old group, of which 7.2% are classified as excellent considering their age, 50.4% as average, and 42.4% as below average.

The incidence of chest pathology is not great even in this group. No disabling cases of silicosis were detected. Minimal degree fibrosis, first degree fibrosis, etc., denote only stages of silicosis and in no way disable the individual from continued employment. Four cases of tuberculosis were diagnosed, one of which was active with a minimal degree of fibrosis associated, the other three healed, no fibrosis associated. A disabling aortic aneurysm was detected in one instance. As in the group of employees not subjected to hazardous occupations the incidence of faulty or im-

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* In *Illinois Medical Journal*, April, 1938.

[1] J. B. HARNEY, M.D., Roentgenologist, Chicago.

paired vision is great and increases rapidly with age; actual percentages in this study are as follows: 3.3% in those between the ages of 17 to 35 years, 12.3% between 36 to 45 years, 13.5% between 46 to 55 years, and 18% between the ages of 55 to 70 years. The condition of the mouth is very poor, dental care being needed in 17.2% of men between the ages of 17 and 35 years, 33.3% between the ages of 36 to 45 years, 34.6% between the ages of 46 to 55 years, and in 32.1% of employees between the ages of 55 to 70 years.

High blood pressure, medically spoken of as hypertension, is known to be a symptom of increasing age, which predisposes to apoplexy, cerebral accidents, or "strokes." Laborious occupations, irregular living, poor oral hygiene, faulty elemination, etc., tend to aggravate this condition. The incidence of hypertension in this study is somewhat great and may be attributed in many instances to one of the previously mentioned facts. The actual percentage incidence below the age of 35 years for all classes of employees, namely 0.95%, is very low as compared to 4.3%, 13.9% and 32.5% in the age groups 36 to 45 years, 46 to 55 years and 56 to 70 years respectively.

The incidence of hernia is not great in this series, being 3.4%. The recording of hernias during a physical examination for occupational disease is not exactly fair to the employee when one considers the hundreds of men employed with hernias who are not exposed to an occupational disease hazard, and although they may perform more strenuous duties, are not examined prior to the commencement of employment.

The presence of varicose veins is characteristic of age, and often this is

TABLE I

Age, Years	Exposure or Occupation	Number of Examinations	Physical Status: Excellent No	Excellent %	Average No	Average %	Below Average No	Below Average %	Questionable No	Questionable %	Chest Findings: Healthy Chests No	Healthy Chests %	Minimal Degree Fibrosis No	Minimal Degree Fibrosis %	First Degree Fibrosis No	First Degree Fibrosis %	Second Degree Fibrosis No	Second Degree Fibrosis %	Tuberculosis No	Tuberculosis %	Other Findings No	Other Findings %
	Non-Hazardous	74	61	82.4	12	16.2	...	....	1	*1.3	74	100	...	....	...	...	...	...	...	.....	...	...
	Battery Plant	8	6	75	2	25	...	....	...	....	8	100	...	....	...	...	...	...	...	.....	...	...
17	Sandblasters	2	2	100	...	....	...	....	...	....	2	100	...	....	...	...	...	...	...	.....	...	...
to	Foundrymen	12	8	75	4	25	...	....	...	....	12	100	...	....	...	...	...	...	1	*8.33	...	...
25	Other*	10	7	70	3	30	...	....	...	....	9	90	1	*10	...	...	...	...	...	.....	...	...
	Non-Hazardous	26	20	77	5	19.2	...	....	1	*3.8	26	100	...	....	...	...	...	...	...	.....	...	...
26	Battery Plant	14	7	50	6	43	1	7	...	....	13	93	...	....	...	...	...	...	(Aneurysm)		7	7
to	Sandblasters	5	2	40	3	60	...	....	...	....	3	50	2	40	...	...	...	...	...	.....	...	...
35	Foundrymen	12	6	50	6	50	...	....	...	....	10	83.3	2	16.7	...	...	...	...	...	.....	...	...
	Other*	47	27	57.8	19	40.5	1	1.7	...	....	44	93.7	3	6.4	...	...	...	...	...	.....	...	...
	Non-Hazardous	55	24	43.6	30	54	1	2.4	...	....	52	94.5	3	5.5	(Not Characteristic)					.....	...	...
36	Battery Plant	7	...	.....	7	100	...	....	...	....	7	100	...	....	...	...	...	...	...	.....	...	...
to	Sandblasters	6	...	.....	6	100	...	....	...	....	5	83.3	1	16.7	...	...	...	...	...	.....	...	...
45	Foundrymen	24	5	20.8	18	75	1	4.2	...	....	23	96	...	....	1	4	...	...	...	.....	...	...
	Other*	44	9	20.5	31	70.5	3	6	1	3	40	90	3	7.5	...	...	1	2.5	...	.....	...	...
	Non-Hazardous	33	8	24.2	23	69.9	2	5.9	...	....	33	100	...	....	...	...	...	...	...	.....	...	...
46	Battery Plant	1	...	.....	1	100	...	....	...	....	1	100	...	....	...	...	...	...	...	.....	...	...
to	Sandblasters	5	...	.....	4	80	...	....	1	*20	3	60	2	40	...	...	...	...	1	*20	...	...
55	Foundrymen	39	2	5.1	27	70	10	24.9	...	....	32	81.6	5	2.7	2	5.1	...	...	2	5.1	...	...
	Other*	36	2	5.5	28	78.1	6	6.4	...	....	33	92	3	8	...	...	...	...	...	.....	...	...
	Non-Hazardous	12	1	8.3	11	91.7	...	....	...	....	12	100	...	....	...	...	...	...	...	.....	...	...
56	Sandblasters	2	...	.....	1	50	1	50	...	....	1	50	1	50	...	...	...	...	...	.....	...	...
to	Foundrymen	13	2	15.4	7	53.9	4	30.7	...	....	13	100	...	....	...	...	...	...	...	.....	...	...
73	Other*	13	...	.....	6	46.2	7	53.8	...	....	12	92.4	...	....	...	...	...	...	...	.....	...	...
	Grand Total	500	199	39.8	260	52	37	7.4	4	.8	468	93.6	26	5.2	3	.6	2	.2	3	.6	1	.4

*Dust Exposure

Technique MA200 KV (Variable Factor) Distance 72 in. Time .1 sec. (one-tenth sec.)

Age, Years	Exposure or Occupation	Number of Examinations	Physical Examination Findings: Poor Vision No	Poor Vision %	Poor Dental Hygiene No	Poor Dental Hygiene %	Heart: Rheumatic No	Rheumatic %	Heart: High Blood Pressure No	High Blood Pressure %	Hernia No	Hernia %	Varicosities No	Varicosities %	Laboratory Findings: Positive Wassermann and Kahn Test No	Positive Wassermann and Kahn Test %	Glycosuria No	Glycosuria %	Albuminuria No	Albuminuria %	Kidney Stone No	Lead Absorption No	Lead Absorption %
	Non-Hazardous	74	2	2.7	4	5.4	...	...	1	1.4	2	2.7	...	....	...	....	...	....	2	2.7	...	...	....
17	Battery Plant	8	...	....	1	12.5	...	...	...	....	...	....	...	....	...	....	1	12.5	...	....	1	2	25
to	Sandblasters	2	...	....	...	....	...	...	...	....	...	....	...	....	...	....	...	....	...	....	...	...	....
25	Foundrymen	12	2	16.6	1	8.3	...	...	...	....	...	....	...	....	*Denotes percentage not significant						...	...	....
	Other*	10	1	10	1	10	...	...	...	....	...	....	...	....							...	...	....
	Non-Hazardous	26	1	3.8	5	19.2	...	...	...	....	1	3.8	...	....	...	....	...	....	...	....	...	...	....
26	Battery Plant	14	1	7	1	7	...	...	...	....	...	....	1	7	...	....	...	....	...	....	...	4	28.5
to	Sandblasters	5	...	...	1	20	...	...	...	....	...	....	...	....	...	....	...	....	...	....	...	...	....
35	Foundrymen	12	1	8.3	2	16.6	...	...	...	....	1	8.3	...	....	...	....	...	....	...	....	...	...	....
	Other*	47	2	4.2	12	26	2	4.2	1	2.1	1	2.1	...	....	1	2.1	...	....	1	2.1	...	...	....
	Non-Hazardous	55	6	10.9	12	21.9	...	...	3	5.4	...	....	2	3.6	...	....	1	1.8	...	....	...	...	....
36	Battery Plant	7	1	14	3	43	...	...	...	....	...	....	...	....	...	....	1	*14	...	....	...	1	14
to	Sandblasters	6	2	33	4	66	...	...	...	....	1	16.7	...	....	...	....	...	....	...	....	...	...	....
45	Foundrymen	24	5	20.8	7	29	...	...	1	4	1	4	2	8	1	4	...	....	...	....	...	...	....
	Other*	44	2	1.5	12	27	...	...	2	1.5	1	2.3	4	3	2	1.5	2	1.5	...	....	...	...	....
	Non-Hazardous	33	8	24.2	10	30.3	...	...	1	3.0	2	6	4	12	1	3	2	6	1	3	...	...	....
46	Battery Plant	1	...	....	...	....	...	...	...	....	...	....	...	....	...	....	...	....	...	....	...	...	....
to	Sandblasters	5	2	40	3	60	...	...	...	....	...	....	...	....	...	....	...	....	...	....	...	...	....
55	Foundrymen	39	4	10.2	12	30.6	1	2.5	10	24.9	...	....	2	5.1	2	5.1	2	5.1	3	4.6	...	...	....
	Other*	36	5	13.9	13	36.2	...	...	5	13.9	1	2.7	3	8.0	2	5.5	...	....	...	11	...	...	....
	Non-Hazardous	12	1	8.3	5	41.5	...	...	1	8.3	2	16.6	1	8.3	...	....	...	....	...	....	...	...	....
56	Sandblaster	2	1	50	1	50	...	...	...	....	1	50	...	....	...	....	...	....	...	....	...	...	....
to	Foundrymen	13	2	15.4	3	23.1	...	...	5	38.5	2	15.4	1	7.7	2	15.4	1	7.7	...	....	...	...	....
73	Other*	13	2	15.4	5	38.5	...	...	7	53.9	1	7.7	2	15.4	...	....	...	....	2	15.4	...	...	....
	Grand Total	500	50	10	118	23.6	3	.6	37	7.4	17	3.4	22	4.4	11	2.2	10	2	13	2.6	...	...	....

*Dust Exposure.

indirectly the cause of prolonged periods of convalescence following mild injuries. The incidence in this series is as follows: 0.48% between the ages of 17 and 35 years, 5.8% between the ages of 36 and 45 years, 7.8% between the ages of 46 and 55 years and 5% between 55 and 70 years of age.

Syphilis, the dread of the poorer classes and a privilege of the wealthy, was detected in 2.2% of cases which is somewhat below the 5% incidence recorded in many studies. This fact is interesting because as previously stated this study included a group of men who under any other circumstances would not have received such a detailed examination.

Ten cases of glycosuria which predisposes to diabetes were detected. This discovery alone is worth the cost of all the examinations.

The significance of albuminuria varies in relation to the case in question. It may denote a low grade infection, a previous night out, or if associated with hypertension a kidney involvement.

TO CULMINATE my talk and to strengthen my plea for routine pre-employment and periodical medical examinations of all employees whether they are exposed to hazardous occupations or not, I wish to point out the following facts:

All conditions as discussed whether directly related to occupation or not can for the most part be prevented, at least improved, provided proper attention is given.

Faulty vision can be corrected, greater work efficiency resulting, less minor accidents occurring, and in all probability improved health of the individual.

Poor oral hygiene predisposes to infections, sinus trouble, rheumatism, arthritis, gastro-intestinal disturbances, faulty elimination, with resulting sluggish habits, poor work efficiency and so on. All of which could be prevented.

High blood pressure characteristic of age and known to be aggravated by dietary indiscretions, often adds unnecessary burden to a silicotic lung. There are relatively few cases of disabling silicosis on record. When silicosis is complicated by a bad heart the clinical symptoms are much more pronounced, likewise when tuberculosis is a complicating factor. It is needless to mention the value of routine medical examination here.

Hernia and varicose veins, conditions which are amenable to treatment, may if they are not taken care of predispose to marked intestinal disorders, slow healing ulcers or even cerebral accidents.

Syphilis is not the dread disease, so considered by many, and should not be the cause of discharge from employment except in its transmittal stage. It is today one of the few diseases for which there is a specific treatment, and an early diagnosis is imperative to cure. Hence the benefit of routine examination in this regard.

Diabetes responds miraculously to therapy, and it seems a pity that all people predisposed should not have the privilege of a routine urinalysis to detect it in its incipiency. The occurrence of one diabetic coma in a small plant with a possible accidental injury associated may entail more cost than a monthly routine urinalysis of all employees for an indefinite period.

One cannot help but assume, hardly conclude, because the series of cases is somewhat small, that the incidence of occupational disease is not great in comparison to other more common every day ailments. Occupational diseases are known to be preventable, modern engineering methods have proved this; likewise, many of our complaints can be prevented if detected early. The conclusion being: *routine pre-employment and periodical examination, the interest of the employer in this aspect of the employee's welfare*, with resulting *work efficiency, fewer accidents, lower insurance cost*, and lastly but not least, *a healthy, robust and good-natured employee.*

Occupational Disease Compensation

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WORKMEN'S compensation insurance is generally considered progressive social legislation.* It is therefore surprising to most people to know that the beginning of such law is traced to a European monarchy, and the author was Prince Von Bismarck, who in the train of social unrest following the Franco-Prussian war, introduced it for the German industrial worker in 1884—some historians say for reasons of political expediency, a sop to a discontended population.[1,2] In 1897 we find this legislation in Great Britain, known as the Workmen's Compensation Act of 1897; and in 1904, even under the regime of oppressive Czaristic government in Russia, provision for compensation for injured workmen was the law of the land.

Not until 1910 was compensation introduced into this country, when New York passed the first law; and 10 states enacted compensation legislation in 1911.[3] These laws of course applied only to traumatic accidents in industry. The New York law was declared unconstitutional, but after a constitutional amendment a new law was passed and became effective in 1914.[3] Today 46 of the 48 states provide for benefits to injured workmen,[4] the only exceptions being Mississippi and Akransas. In late years these laws have been generally extended to include recognized diseases which are inherent in many industrial and manufacturing processes.

There are two general plans used by various states in providing these benefits: the so-called "schedule," which is an enumeration of such occupational diseases as the law contemplates should be compensated, and the so-called "all-inclusive plan," which may extend benefits to many diseases and conditions not contemplated under the "schedule" plan. Most states have provided coverage for occupational diseases by an extension of their existing traumatic laws. A very few within the last two or three years have enacted separate compensation legislation affecting occupational disease hazards distinctly apart from the traumatic acts. Among these are the states of Illinois and Indiana, whose occupational disease laws are substantially alike, the Illinois law having become effective in 1936 and the Indiana law in 1937.

Outstanding among the states having scheduled occupational-disease laws are New York, Minnesota, and Ohio. The scheduled laws generally name from 20 to 30 occupational diseases which are inherent in common manufacturing processes. The United States Department of Labor lists 63 principal industrial diseases found in this country.

The ones most commonly known to the public are silicosis, found in the stone and foundry industries; lead poisoning; conditions resulting from exposure to chemicals and acids of many kinds; and dermatitis, of which there are many varieties. Even such conditions as writers' cramp and frost-bite are included in some jurisdictions.

The old Indiana traumatic law provided that compensation should not include disease except as the result of injury. Labor leaders and social reformers have long agitated for benefits to workmen contracting diseases in industry, and occupational-disease legislation is becoming rapidly extended throughout the country. In addition to Indiana, the states of Delaware, Illinois, Michigan, Ohio, Pennsylvania, Nebraska, Washington, and Wisconsin adopted these laws or amendments of existing laws during their 1937 legislative sessions. This makes 21 states and the District of Columbia which compensate for occupational diseases.[4]

An examination of the provisions of the Indiana law will give some idea as to how this type of legislation protects the disabled employee. The Indiana law provides that the act shall be elective, and election is accom-

* Condensed from paper presented before the meeting of the Indiana State Nurses' Association, Terre Haute, Indiana, October 11, 1937; published as here in *Public Health Nursing*, May, 1938.

plished by the employer filing a written acceptance of the act with the industrial board.* If he does not file a written acceptance, or election, to be subject to this law, he remains subject to common law action by the complaining employee with the common law defenses: (1) assumption of risk (2) negligence of fellow-servant (3) contributory negligence, removed. The only common law limitation of liability is in the event of death, where the statutory limit of $10,000 applies.

An employer having elected to operate within the provisions of the law has, for a period of four years from 1937, the option of recalling his acceptance by filing notice with the industrial board 60 days prior to October 1 of any of these years. He may again elect to come under the law, but upon second acceptance he is permanently bound by its provisions. The occupations specifically excluded from the application of the law are: (1) casual laborers (2) farm or agricultural employees (3) domestic servants (4) employees in railroad train service. The definitions of what shall constitute an occupational disease seem ambiguous and difficult of interpretation. As has been true in Illinois, there will probably be in Indiana a considerable number of appeals to the high courts for interpretations as to occupational diseases and application of this law.

Briefly, the principal benefits provided for are as follows: in the case of death, 55% of the average weekly wage, for a maximum period of 300 weeks, plus $100 burial expense; for temporary total disability, 55% of the weekly wage, for a maximum period of 500 weeks; for temporary partial disablement, 55% of the difference between the average weekly wage before and after disablement, not to exceed 300 weeks. There is in this law, as in the traumatic act, a waiting period of seven days before payments begin after disability. Disablement in order to be compensable must occur within one year of the last day of last exposure to the hazards of the disease, except in cases of silicosis or asbestosis, when the limit is extended to three years. In the case of death, it is compensable only if it occurs within one year after date of disablement — except where there is a pending claim—and in no event, more than 300 weeks after disablement. The law gives a schedule of benefits for specific disablements, for amputations, loss of use of member, and total or partial total permanent disabilities. Provision is made for disfigurement in the discretion of the industrial board, but with benefits not exceeding 200 weeks. As in the traumatic act the maximum weekly compensation is $16.50; minimum $8.80.

Limitation of filing of claim is two years after the last day of the last exposure and within one year from date of disability in case of disablement; in the case of death, action must be commenced within one year after death and within three years after the last day of the last exposure. The law provides that there shall be a presumption of conclusive liability, regardless of length of exposure, upon the last employer, except again in the cases of silicosis and asbestosis, when the employer liable is the one in whose employment the employee was last exposed during a period of 60 days or more. The rules of administration and provisions for insurance and procedure follow closely, if not exactly, those applying to the old traumatic law.

The part of the nurse in the industrial program is obvious. The early recognition of symptoms indicating disease is important. A thorough understanding of these diseases, with an intelligent program of prevention is more important. Occupational diseases are preventable and as susceptable to control as are accidents—perhaps even more so. A healthful environment, safe industrial processes, and the education of employees to the importance of safety are main features of control. In many cases the substitution of materials is practical as a means of preventing hazards. All of this is a fertile field in the conservation of health and life—a worthwhile investment in elimination of human wastage in industry.

Ohio Medical Referees

AS EMPOWERED by Sub. House Bill 71 enacted last year by the Ninety-Second Ohio General Assembly, adding silicosis to the list of occupational diseases compensable under the Ohio Workmen's Compensation Act, the State Industrial Commission has appointed the following physicians as silicosis referees: Dr. Raymond C. McKay, Cleveland; Dr. Joseph A. Muenzer, Toledo, and Dr. William M. Doughty, Cincinnati.*

In selecting the referees, the Commission followed the provisions of the Act that they "shall be licensed physicians in good professional standing, who have by special duty, or experience, or both, acquired special knowledge of pulmonary diseases and at least one of said physicians shall be a roentgenologist." All silicosis claims must be passed on by the referees.

* *Ohio State Medical Journal*, May, 1938.

Brief biographical data concerning the appointees, all of whom have had clinical experience with silicosis, follows:

Dr. McKay graduated at Western Reserve University School of Medicine, Cleveland, in 1920. He is in active charge of the tuberculosis unit of Cleveland City Hospital and is assistant department head of the medical staff of that unit. Dr. McKay also is assistant clinical professor of medicine at Western Reserve University. He is a member of the Ohio State Medical Association, Fellow of the American Medical Association, member of the National Tuberculosis Association, the American Sanatorium Association and the American Academy of Tuberculosis Physicians.

A graduate of St. Louis University School of Medicine in 1918, Dr. Muenzer served in the Navy Medical Corps from July, 1918, to August, 1919. From 1919 to 1925, Dr. Muenzer was on the staff of the medical dispensary at St. Vincent's Hospital and at Lucas County Tuberculosis Hospital. From 1921 to 1931 he did diagnostic work for the Bureau of Tuberculosis, State Department of Health. Dr. Muenzer has done experimental clinical research in endocrinology at St. Anthony's Orphanage. He is a member of the Ohio State Medical Association and the A.M.A.

Dr. William M. Doughty is director of radiology at Christ Hospital and Children's Hospital, Cincinnati. He was formerly assistant director of roentgenology at Cincinnati General Hospital and formerly associate professor of radiology at the University of Cincinnati, College of Medicine, which positions he resigned to become a member of the Board of Directors, University of Cincinnati. A graduate of Miami Medical College, Cincinnati, in 1906, Dr. Doughty served an internship of 18 months in Cincinnati General Hospital and did postgraduate work in London and Vienna for one year. He is a past-president of the Cincinnati Academy of Medicine; member of the Ohio State Medical Association; Fellow of the American Medical Association, American College of Surgeons and the American College of Roentgenology; member of the American Roentgen Ray Society and the Radiological Society of North America.

DR. McKAY

DR. MUENZER

DR. DOUGHTY

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Reasonable compensation of the silicosis referees and of other physicians used and the expenses of examination and tests will be paid, if the claim is allowed, as part of the expenses of the claim; otherwise from the surplus fund of the Commission.

Silicosis is defined in the Act as: "A disease of the lungs caused by breathing silica dust (silicon dioxide), producing fibrous nodules, distributed through the lungs and demonstrated by x-ray examination or by autopsy."

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